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NEUROSES

MEDICAL AND SURGICAL THERAPY

EDITED BY

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VOLUME II

NEUROSES

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NEUROSES

CLINICAL FORMS OF LESIONS OF NERVES

TREATMENT AND REPAIR OF NERVE LESIONS

HYSTERIA OR PITHIATISM

THE PSYCHONEUROSES OF WAR

BY

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BY

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BY

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SECTION I

CLINICAL FORMS OF NERVE LESIONS

Clinical Forms of Nerve Lesions

CHAPTER I

METHOD OF EXAMINATION

For the examination of lesions of the peripheral nerves rapidity must be combined with a thoroughness which will obviate any fear of overlooking signs helpful to an accurate diagnosis.

I. Previous History

The previous history should be inquired into for the following particulars :—

A. **Details of the Injury.**—Date of wound. Nature of projectile. Localisation of the different wounds (if any).

Posture of the patient when he was wounded. The posture may account for some nerves, ligaments, and vessels being affected which, on first inspection, appeared to be outside the course of the projectile.

Sensations felt by the wounded at the time of injury : pain, numbness, stabbing, electric current. The locality and radiation of such sensations.

B. **Immediate Results.**—Was there *loss* of *consciousness* or collapse ? Was there any other traumatism associated with the injury, due to contusion or falls ? On what part of the body were these secondary blows inflicted ?

Involuntary movements made by the injured limb immediately after penetration of the projectile. Spasmodic contractions of certain muscles, tremors, etc.

Was the paralysis immediate or progressive? If the latter, how long after injury did the first symptoms appear?

Did the *patient lose much blood* immediately? Was a tourniquet applied, or too tight a dressing, and *for how long*? What was the condition of the limb, and particularly of the hand or foot, after this constriction (discoloration, œdema, lowering of local temperature, etc.)?

Was an operation undertaken immediately? On which tissues? Were foreign bodies or bony fragments extracted? Was an *artery ligatured*? Or a *nerve sutured or freed*, etc.? Was the lung injured (hæmoptysis, subcutaneous pulmonary emphysema)?

Was antitetanic serum injected?

Was there *secondary hæmorrhage* within a few hours or days of the injury?

Was there a *fracture*? What was the position of the limb after injury, and what appliances were used (slings, plasters, splints, etc.)?

C. **Subsequent History.** — *Secondary hæmorrhage. Suppuration.* Serious infection (gas gangrene, tetanus, etc.). *Necessary surgical intervention* on bones and *nerves.* Duration of suppuration. Duration of consolidation of the fracture (if any).

Evolution of motor disturbances.—Has the paralysis retroceded, increased, or remained stationary?

Evolution of sensory disturbances:—

Subjective disturbances.—Pain, paræsthesia, burning sensations: date of their appearance, increase or diminution, their radiation and changes in character.

Objective disturbances. — Anæsthesia, hyp- and hyperæsthesia.

Evolution of trophic, vasomotor, and secretory disturbances.—Progress of muscular atrophy: diminution

or increase of vasomotor phenomena (colour of the skin, temperature of the limb). Secretory troubles (perspiration). Appearance of sloughs and ulceration. Alterations in the appearance and growth of the nails and hair. Condition of the skin.

Evolution of articular disturbances. — Increase or diminution of ankylosis, and their causes.

Methods of treatment for paralysis or impotence of the limb, with results. Has the patient used crutches or sticks ?

The results of skiagraphy (foreign bodies, callus formation, etc.).

II. Examination of the Patient

The appearance alone of an injured limb (dropped wrist, clawing of the fingers, deformities) will indicate to the experienced observer to which muscles he should turn his attention, and the investigations he should make ; but a systematic and thorough examination is indispensable to obviate surprises and mistakes.

ARTICULATIONS

In order to form a correct estimate of the extent of the motor disturbances before investigating the so-called nerve lesions, the mobility and range of movement of the joints should be examined by comparing them with those of the sound side.

A. **Upper Limb: Shoulder.** — Tell the patient to raise his injured arm and to put his hand on his head. Carefully watch and palpate the scapula during this movement, and observe how soon it becomes displaced. Note the height to which the arm can be raised. Test the internal and external rotation of the humerus, and ask the patient to put the affected arm behind his back with the hand on the opposite buttock : then to cross his arms, putting each hand on the opposite shoulder.

Elbow.—The range of extension and flexion should be noted. The movements of pronation and supina-

tion should be tested by the observer holding the patient's elbow firmly in his left hand, and with his right turning the wrist backward and forward.

Wrist.—The range of extension, flexion, and of lateral movements (abduction and adduction) should be ascertained.

Fingers.—The amount of abduction and adduction of the thumb and fingers should be tested. The articulations of the phalanges should be most minutely examined and compared with those of the sound hand.

B. **Lower Limb: Hip.**—The movements of flexion, extension, adduction, abduction, and rotation should be examined, and it should be ascertained whether the two iliac spines are on the same horizontal level and whether a lumbar curvature or a scoliotic deformity of the vertebral column is present.

Knee.—The range of extension and flexion should be tested.

Ankle.—Extension, flexion, abduction, and adduction should be investigated.

CICATRICES AND CALLOSITIES

Attention will be directed at once to cicatrices: scars of entry and of exit, or of operations for the extraction of foreign bodies (these should be accurately entered on a diagram), scars left by incision into areas of inflammation, or by other surgical operations on bones, arteries, or nerves.

Note the adherence of scars to deeper tissues, the induration of surrounding parts and their condition whilst active or passive movements are being made (retraction, depression, etc.).

Ascertain, if possible, their depth in relation to muscles, and pay special attention to cicatrices involving tendons.

In cases of fracture, the density, bulk, and possible pressure of callus on nerve-trunks or vessels, should be investigated by palpation.

MOTOR FUNCTION

Each case requires a special examination, which will be described in the chapters dealing with the different nerve lesions.

But it is important in every case, whatever the level of the injury, to examine the whole of the affected limb from its origin to its extremity, noting the movements which appear weak, imperfect, or impossible. A mere superficial examination would fail to recognise important disturbances of motion.

When a nerve lesion is suspected, and when the course of the missile, the appearance of the limb, and the loss of power complained of by the patient indicate its site, each of the muscles supplied by the nerve under suspicion should be examined.

In making this examination test the passive and active movements at each joint, and compare the action and play of the muscles involved with those of their opponents.

Since Duchenne, of Boulogne, pointed it out, the importance of the part played by the antagonists in the normal function of a muscle, in relation to its energy and effectiveness, has been generally recognised. The tension of the aponeuroses covering the limb when a movement is being made should be taken into consideration.

In testing the contraction of a muscle, both the muscle itself and its tendon should be palpated, especially if it has been subjected to direct injury or to induration resulting from suppuration, of long standing, in the neighbouring tissues. Electrical examination, to be described later, will frequently be of service in ascertaining whether the muscle itself, the tendon, or the motor nerve has been injured.

MUSCULAR ATROPHY

When testing the activity of a muscle it is most important to estimate, if necessary by measurements, the amount of atrophy present. This information

is valuable in diagnosing the gravity of the nervous lesion.

SENSORY FUNCTION

Objective Disturbances.—Cutaneous or superficial sensibility and deep sensibility should each be studied in turn.

A. **Superficial Sensibility** includes :

1. Sensibility to touch.
2. Sensibility to pain.
3. Sensibility to heat and cold.

1. Examination for tactile sensibility should be made with very light stimulants so as to avoid arousing deep sensations ; for this purpose a wisp of cotton-wool or a small, soft camel-hair brush is suitable.

The segment of limb under examination should be supported on a horizontal plane and not held by the examiner, and the patient should be instructed to relax his muscles as much as possible. He should be blindfolded and asked to say " Yes " whenever he feels contact.

The area of anæsthesia, where the touch is not felt at all, should be defined with a dermographic pencil, and then the area of hypæsthesia, where tactile sensibility is only diminished or retarded, should be mapped out in a similar manner.

To avoid possible error and to test the patient's attention, the examiner from time to time should cease making contacts and see if he still answers in the affirmative, or else ask the patient to indicate the spot where he has been touched.

The examination of sensory disturbances should be repeated on several occasions, as the subject's attention always tires appreciably after a time, which varies with the individual and his condition of health.

It is better to work from those parts supposed to be sound to the areas in which tactile sensibility is diminished.

Having determined the existence and limits of anæsthesia and hypæsthesia, errors of localisation in the latter region should be checked by requiring the patient to indicate the spots where he is touched. It is in the region of hypæsthesia that errors in tactile discrimination can also be detected. For this purpose a pair of compasses with blunt points should be used.

After separating the points of the compass to the desired extent, contact with the skin should be made irregularly, first with one point, then with both, and the patient should respond "One" or "Two" whichever he feels.

This power of feeling two touches made simultaneously is usually found to be diminished in nearly the whole of the region of tactile hypæsthesia, the patient only recognising the double touch when the points of the compasses are wide apart.

2. *Sensibility to pain* should be tested with a pin. As in the case of tactile sensibility, an area of painful anæsthesia, or analgesia, as extensive as that of tactile anæsthesia will be found always surrounded by a zone, varying in extent, of painful hypæsthesia, where the pin-prick is felt as a touch, as a dull, diffused pain, or only appreciated after an interval of delay. In other cases, again, there is neither analgesia nor hypalgesia, but hyperalgesia, which may coincide with diminution or absence of superficial tactile sensibility.[1]

3. *Sensibility to heat and cold* can be adequately tested by using ordinary test-tubes, of a diameter of at least a centimetre, filled with hot or cold water and containing a thermometer. Metal tubes, being better conductors of heat, are preferable. The hot

[1] Painful sensibility can also be tested by faradisation, using as the active electrode a Duchenne's brush attached to the negative pole of the battery. M. E. Carati, of Bologna, who has made this subject his special study (*Revue Neur.*, May–June 1915), comes to the conclusion that, tested by faradisation, the areas of variation in painful sensibility are always more extensive than the areas of hypæsthesia, anæsthesia, or hyperæsthesia arrived at by other methods.

water used should not be of a higher temperature than 50° C.: the cold, by adding crushed ice, should be kept at about 0°.

Thus a complete area of thermal anæsthesia, as extensive generally as the area of tactile anæsthesia, is often found surrounded by a more or less narrow border where temperatures of 50° and 0° are only appreciated with delay, and sometimes wrongly interpreted, and where the intermediate temperatures, between 20° and 40° C., are either not recognised or only recognised with difficulty.

Often, in this area, the application of a tube of hot water at a temperature of 48°–50°, at first perceived as a simple touch, will presently produce a painful sensation of persistent and diffuse burning.

B. **Deep Sensibility** is of three varieties:

1. Sensibility to pressure.
2. Sense of passive movement and posture.
3. Sensibility of bones.

1. *Sensibility to pressure*, or *barœsthesia* or the sensation of weight, may be tested by either the tip of the finger or the head of a pin. In some cases within an area where superficial sensibility to a flake of cotton-wool or to a fine camel-hair brush is abolished, pressure with the head of a pin or the tip of the finger is readily appreciated and correctly localised.

2. *Sense of passive movement and posture* should be investigated in the following manner:—The patient's eyes should be bandaged, and after making a number of passive movements on a segment of the affected limb (fingers, toes, wrist, ankle), the examiner should ask the patient to carry out the same movements with the sound limb, or else to clearly describe them.

After performing a variety of passive movements, a segment of the limb should be placed in a given posture (flexion, extension, abduction, adduction), and, taking care that the muscles are completely relaxed during the investigation, the patient should be asked to describe the position of the segment and told to

place the corresponding segment of the sound limb in the same posture.

3. *Osseous sensibility* should be tested with a large tuning-fork producing powerful vibrations.

After making the fork vibrate by striking it (*e.g.* with a reflex hammer), its base should be placed on the surface of the bone under examination, selecting spots where the bony surface lies close beneath the skin.

Stereognosis.—Having completed the tests of superficial and deep sensibility, the faculty of recognising the physical properties of objects may be investigated.

This power of appreciating the size, form, consistence, and nature of objects is often affected in lesions of the peripheral nerves. It can be tested by allowing the patient, when blindfold, to feel different things, such as a watch, key, pencil, coin, button, etc.

This particular sense or faculty is dependent on a combination of simple sensations conveyed at one and the same time by the paths of superficial and deep sensibility. It frequently happens, when motor paralysis does not entirely prevent palpation of an object, that the patient will recognise some qualities, such as shape or consistence (primary identification), without being able to name the object itself (secondary identification).

The more important the sensory distribution of the injured nerve, whether superficial or deep, the more the faculty of recognising the physical properties of objects is affected.

Subjective Disturbances are always difficult to estimate, the details given by the patients varying with their degree of intelligence and their inclination to complain. They should be questioned about *formication and numbness*, and particularly whether pain, if present, is constant or paroxysmal, localised or diffuse. The nature of the pain, too, should be carefully noted, whether smarting, burning, shooting, pricking, or grinding, as well as its seat, along the course of a nerve or in a definite sensory area, such as the palm of the hand or sole of the foot.

It is equally important to ascertain whether the pain is *spontaneous* or *provoked* by some act such as pressure on the nerve or its terminal expansions. Note also any conditions which increase or diminish its intensity.

REFLEXES

The tendon, bone, and periosteal reflexes should be examined with a reflex hammer.

The muscles of the limb should be completely relaxed, and each reflex should be investigated several times. The corresponding limbs should always be examined symmetrically to realise any difference in the reflex response.

Upper Extremity:—

Tendon reflexes.—The chief reflex is the *tricipital*, which is obtained by striking the tendon of the triceps just above its insertion into the *olecranon*. The response is an extension of the forearm at the elbow.

To obtain the necessary, though difficult, relaxation of the muscles, it is advisable to allow the arm to hang quite loose by the side of the body and then to flex the elbow to a right angle, reminding the patient not to stiffen his muscles.

Other reflexes to be tested are those of the biceps, extensors of the wrist, extensors of the fingers, flexor carpi radialis, and flexors of the fingers by percussing their respective tendons at the back and front of the wrist.

Osteo-periosteal reflexes.—The *stylo-radial reflex*, obtained by tapping the stylo-radial process. Normally this evokes a contraction of the biceps, the brachialis anticus, the supinator longus, and even the anterior fibres of the deltoid. To obtain this reflex the wrist must be thoroughly relaxed, the hand placed half way between pronation and supination, and the elbow semi-flexed.

The *ulnar pronator reflex* (Pierre Marie), evoked by

tapping the posterior surface of the styloid process of the ulna, the hand being placed in the position previously described, produces a movement of pronation at the wrist.

The *olecranon reflex*, obtained by tapping the posterior surface of the olecranon below the insertion of the triceps tendon : as opposed to the triceps reflex, it causes a movement of flexion of the forearm on the arm.

Finally, by tapping the vertebral border of the scapula a contraction of the pectoralis major, of the deltoid and even of the other shoulder muscles may be obtained.

There are no important cutaneous reflexes of the upper extremity.

Lower Extremity:—

Tendon reflexes.—The two most important reflexes are the *patellar reflex or knee-jerk*, and the *tendo Achillis jerk*.

The *patellar reflex*, obtained by tapping the patellar tendon, is a movement of extension of the leg on the thigh, and is elicited with the knee in a position of semi-flexion. If the patient is lying down the popliteal space should rest on the arm of the observer ; but if he is sitting, his feet should be placed flat on the ground, the leg with the thigh making a slightly obtuse angle. The patient can also be examined seated on the edge of his bed with his legs hanging down. The observer's left hand should always be applied to the anterior aspect of the thigh in order to observe the slightest contraction of the quadriceps extensor femoris.

The *tendo Achillis* jerk is obtained by striking the tendo Achillis tendon with the patient kneeling on a stool or a chair with the muscles of his foot completely relaxed. The gastrocnemius contracts and causes plantar flexion of the foot. The foot should be held at a right angle with the left hand in order to detect the slightest movement and to avoid

mistaking the motion of the whole foot produced by percussion for the reflex contraction.

The other tendon reflexes of the lower extremity are less important—reflexes of the tibialis anticus, tibialis porticus, and peronei obtainable by percussion of their respective tendons at the ankle.

Osteo-periosteal reflexes are of less significance than those of the upper extremity ; the chief is the so-called *Mendel-Bechterew reflex*, which evokes flexion of the four last toes on percussion of the bone at the angle formed by the os calcis and astragalus. *Adductor reflexes* are obtained by percussion on the internal condyle of the corresponding femur.

Cutaneous reflexes in the lower extremity are of extreme importance.

The *cutaneous plantar reflex* is obtained by stroking the outer edge of the sole of the foot with the point or head of a pin from the heel to the toes. When friction of the outer edge is ineffectual, the middle of the sole of the foot, or even its inner side, should be stroked before assuming the reflex to be abolished.

Normally, the effect produced will be plantar flexion of the toes ; but when there is a lesion of the pyramidal tract, extension or dorsal flexion of the great toe results. This is known as *Babinski's sign*, and may or may not be accompanied by extension or separation of the other toes producing the "fan sign" of Dupré.

When there is no response to plantar stimulation, sharp friction of the inner aspect of the tibia (*Oppenheim's sign*), pinching the tendo Achillis, or pressure on the muscles of the calf may be tried.

Reflex of the fascia lata (Brissaud) is also obtained by friction of the sole of the foot, and is manifested by a visible contraction in the upper and outer aspect of the thigh, which usually accompanies the toe movements produced by plantar stimulation.

Reflex adduction of the foot is produced by scratching the inner edge of the foot at the junction of the plantar

and dorsal aspects. It may be present independently of Babinski's sign, and is frequently observed after lesions of the cerebral cortex (Pierre Marie and Henry Meige[1]).

The *cremasteric reflex* is obtained by pinching the skin on the inner aspect of the thigh, or, better still, by stroking its inner aspect with a pin from the groin towards the knee; in a male patient, retraction of one or both testicles towards the inguinal canals may be produced.

Of minor importance is the *gluteal reflex* obtained by stimulating the skin over the buttock, and manifested by a contraction of the glutei.

It is advantageous, too, to test two other cutaneous reflexes—the *abdominal reflexes* (superior, median, and inferior), consisting of a sharp contraction of the muscles of the abdominal wall when the skin of the abdomen is stroked from the margin of the ribs downward. The patient should be recumbent and the muscles of the abdominal wall quite relaxed.

The *anal reflex*, a contraction of the sphincter ani, is best elicited by placing the patient in the genu-pectoral position, or flat on his abdomen with thighs wide apart, and pricking the perineum about an inch from the margin of the anus.

IDIOMUSCULAR CONTRACTION

Idiomuscular contraction is obtained by striking the mass of a muscle itself with a reflex hammer, preferably at the spot where the nerve penetrates the muscle, *i.e.* its motor point.

Sicard and Cantaloube[2] have recently indicated the best points for stimulating the small muscles of the hand and foot. In the hand the *thenar* muscles should be percussed near their centre (flexion and opposition of the thumb).

[1] Pierre Marie and Henry Meige, "Le réflexe d'adduction du pied," *Soc. de Neurologie*, March 2, 1916.

[2] Sicard and Cantaloube, "Les réflexes musculaires de la main et du pied," *Presse médicale*, April 3, 1916.

The *hypothenar* on the inner and posterior part of the hypothenar eminence (flexion and abduction of the little finger).

The *interosseous* in the dorsal intermetacarpal spaces, particularly the first. Adduction of the fingers results.

For the foot, the *extensor brevis digitorum* should be tested by tapping the outer aspect of the foot at the level of the junction of the os calcis and astragalus (extension of the second and third toes). The *interosseous* muscles of the foot, when tapped on the dorsal surface of the foot at the proximal end of the intermetatarsal spaces of the second and third toes, cause flexion of these toes.

VASOMOTOR, SECRETORY, TROPHIC, AND CALORIC DISORDERS

Alterations in colour of the skin of the whole or part of a limb should be carefully noted and compared with that of the sound limb.

The *temperature* of the limb should be observed, either by simple palpation or by means of a surface thermometer, care being taken that the instrument registers the same level at the beginning of the observation and is kept in contact with the skin for the same length of time when comparing the injured with the sound side.

The *absence* or *hypersecretion of sweat*, the presence of eruptions or ulcers, deformities of the nails, alterations in the growth of the nails and hair, and atrophy or thickening of the skin, should be noted with care.

Important vasomotor, trophic, and caloric disorders, in the great majority of cases, indicate a vascular disorder complicating the nervous lesion.

It is essential, therefore, to feel the pulse and to measure the arterial blood pressure with an ordinary sphygmomanometer (Pachon, Vaquez, etc.).

Electro-diagnosis

(By Dr M. Grunspan)

In the case of neuromuscular lesions an electrical examination is an important aid to clinical investigation : it dispels doubt in complicated and difficult cases, helps in forming a prognosis, and gives valuable indications as to treatment.

No complicated installation is required for this investigation.

An induction coil with two dry cells on the one hand, and a battery of thirty-two elements capable of giving a current of twenty to thirty milliampères, provided with a Courtade key, on the other hand, will be all that is required in most cases. Those who are unaccustomed to electro-diagnosis will meet with some difficulties.

We shall describe briefly the method of procedure : All the apparatus should first be placed within the operator's reach ; the part that is to be examined should be brought into a good light, and the patient placed in such a position that the muscles are thoroughly relaxed ; it is usually better to have him recumbent on a bed.

Investigation should be made both by the unipolar and the bipolar methods, the former alone being inadequate.

In employing the *unipolar* method, a large indifferent electrode is applied to the patient's back between the scapulæ or in the lumbar region, and the muscles and nerves are tested by means of a smaller electrode.

It is frequently necessary, however, to employ strong currents in order to excite contractions in a diseased muscle. The current overflows into the surrounding muscles and the limb twitches violently, with the result that feeble contractions of the affected muscle are completely masked.

It is advisable, therefore, to localise the effects of the current by the use of two testing electrodes

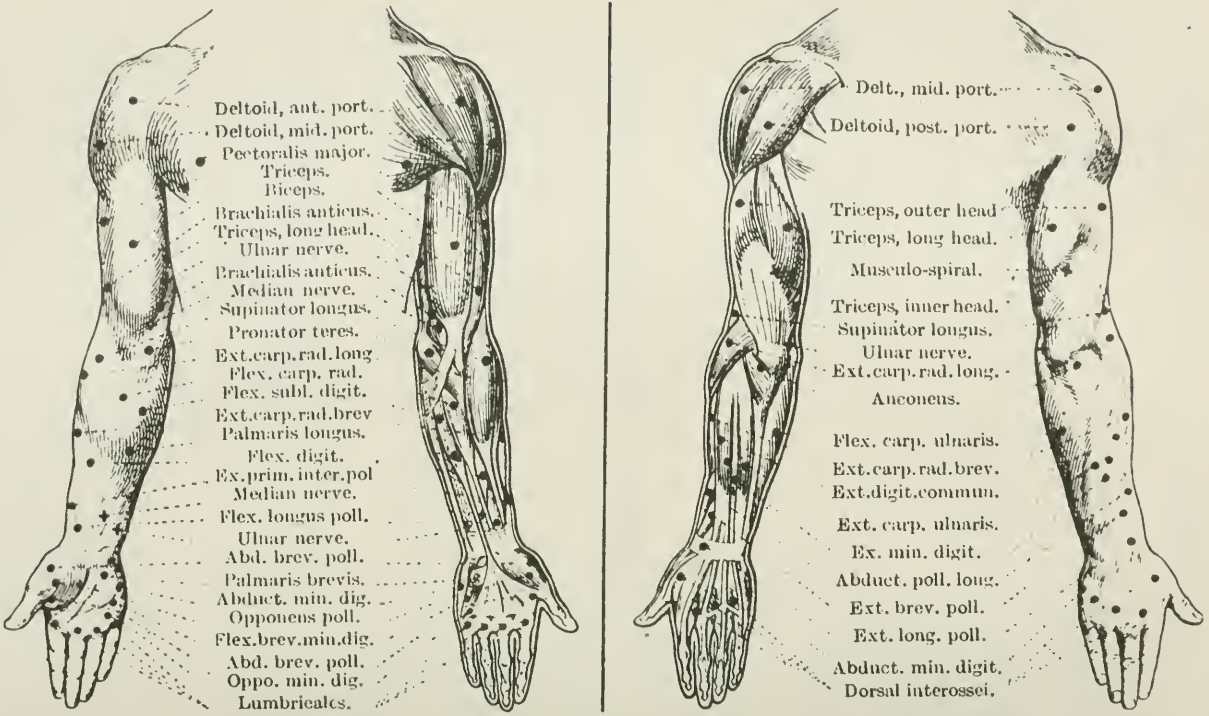


FIG. 1.—Motor points of the upper extremity.

applied to the muscle itself. This constitutes the *bipolar method.*

Faradic Exploration. — In unilateral lesions the excitability of the sound side should be tested first and compared with that of the affected side. The test should be applied to the muscles and nerves consecutively, and the operator should previously have learnt to recognise the different motor points. The nerves can only be tested at those points in their course where they are superficially situated just under the skin. The muscles can be excited in any part of their length, but each displays a very limited zone which responds to the current much more promptly and rapidly than the rest of the muscle. (See figs. 1, 2, and 3.)

The reactions which occur should be noted consecutively. If the muscles and nerves of the affected side respond like those on the sound side, the reactions are said to be normal; but if the response differs from that of the sound side, the reactions are abnormal. These abnormalities may be quantitative or qualitative, or both.

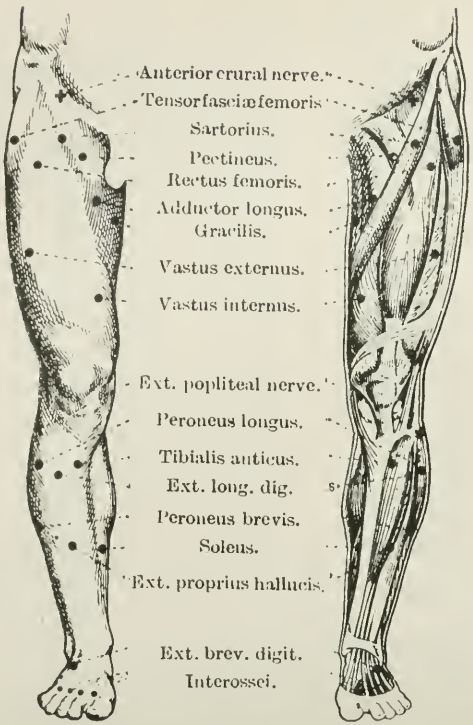
If it is necessary to use an induced current of greater strength to obtain the first sign of contractility in a muscle on the affected side than is necessary to excite a similar contraction in the corresponding muscle on the sound side, *faradic irritability* is said to be *decreased.*

If, on the other hand, the strength of current necessary to excite a minimum contraction in the muscle on the sound side causes a violent twitch on the diseased side, *faradic irritability* is said to be *increased.*

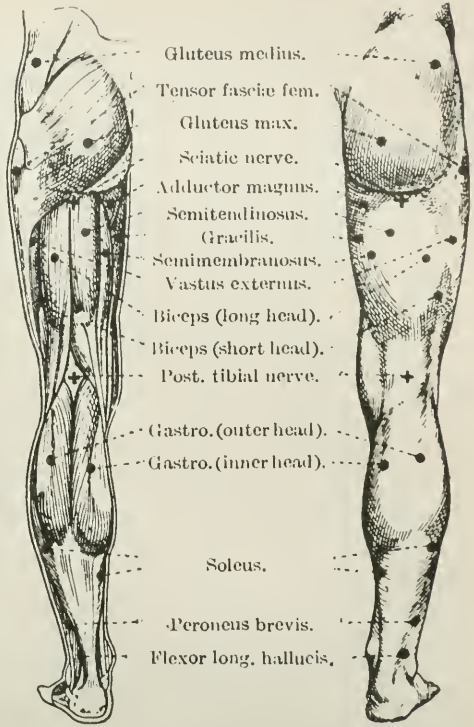
The induction coil may be used at its maximum strength, and no reaction to the test result: there is then said to be *abolition of faradic irritability.*

Galvanic Exploration.—The same principles apply to tests with the battery current: there may be *decrease*, *increase*, or *abolition of galvanic irritability.*

While in faradic examination little or no notice is taken of polar distribution, in galvanic unipolar



(Anterior aspect.)



(Posterior aspect.)

FIG. 2—Motor points of lower extremity.

testing, on the contrary, it is necessary to attach the testing electrode to the negative pole if it is desired to investigate what is called the "inverted formula."

In a normal muscle a greater strength of current is required to obtain a contraction with the anodal closure current than is necessary with the kathodal closure current. If the contrary obtains, or the reaction with both poles is alike, there is said to be an inversion of the polar formula, or polar equality.

A normal muscle responds to galvanic stimulation by contracting like a flash of lightning; when the response is slow, wormlike, and diffuse, the *contraction is said to be sluggish.* This type of contraction is of paramount importance in electro-diagnosis, and is the chief *characteristic of the reaction of degeneration.*

Reaction of degeneration may be either *complete* or *partial.*

Complete reaction of degeneration is indicated *in the nerve* by—

Faradic and galvanic irritability being abolished.

In the muscle by—

1. Faradic irritability being abolished.
2. Galvanic irritability being increased or decreased.
3. (*a*) Response to galvanic stimulation is sluggish; (*b*) normal formula; polar equality or the inverted formula.

When the lesions are less marked, the reactions are not so clearly defined, and the reaction of degeneration will then be *partial.*

Partial reaction of degeneration presents the following features:—

In the nerve—

Decreased faradic and galvanic irritability.

In the muscle—

1. Decreased faradic irritability.
2. Decreased or increased galvanic irritability.
3. (*a*) Sluggish contraction; (*b*) normal formula; polar equality or the inverted formula.

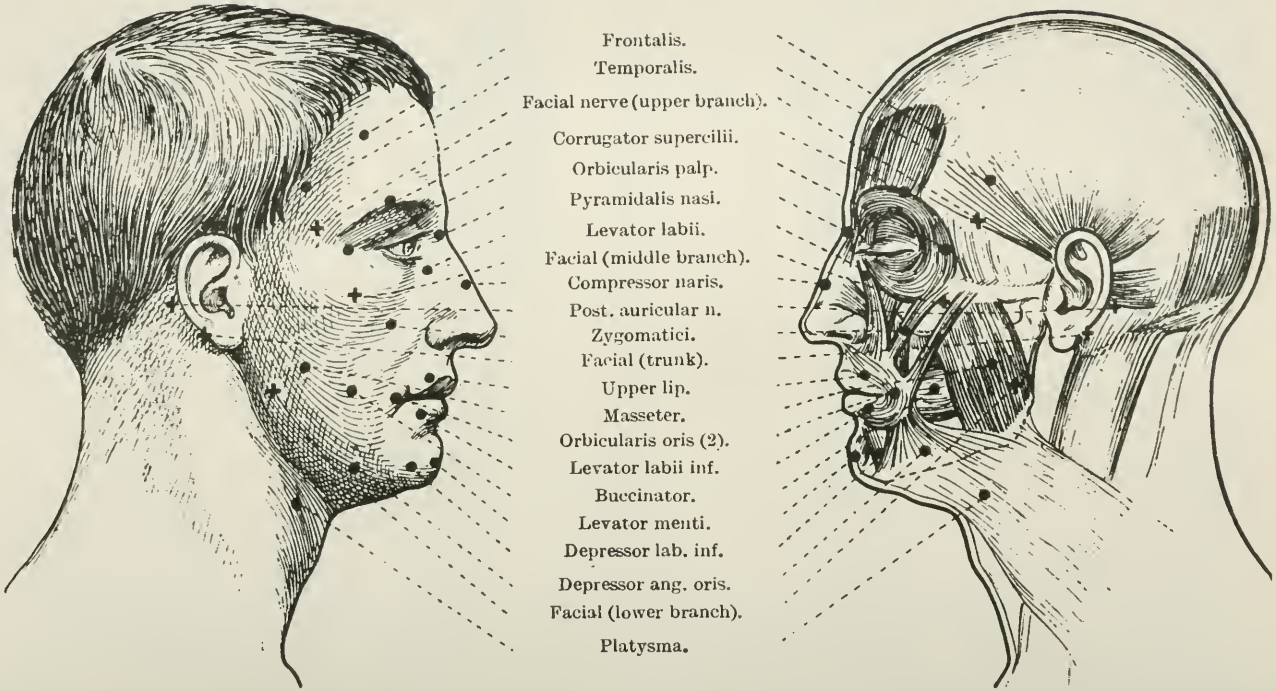


FIG. 3.—Motor points of face.

Text-books attach an importance to the inverted formula which is quite unsupported by experience. It is now known to be of little value, and we regard slowness of contraction as the only valuable evidence of degenerated muscle.

Huet and Bourguignon, Laquerrière and Delherm, Delherm and M. Grunspan have all demonstrated that the myotonic reaction should be incorporated in the reaction of degeneration. This myotonic reaction has hitherto only been observed in Thomsen's disease and in the myopathies. But this reaction occurs in a peculiar manner in the reaction of degeneration: it always seems to disappear at the opening of the circuit, and may be observed both in complete and partial reaction of degeneration.

Illustrations

It is essential to record in graphic form the anatomical and physiological functions of the different nerves.

To the *upper extremity* belong—

the *median* nerve, drawn in dead black.

the *musculo-spiral* nerve, drawn in fine close lines from r. to l.

the *musculo-cutaneous* nerve, drawn in wider lines from l. to r.

the *ulnar*, drawn in heavy lines from r. to l.

the *circumflex*, dotted black

The lower extremity.—The diagrams will be used found on p. 159.

The figures giving the various clinical aspects of the extremities after nerve injuries were drawn from nature and most ably carried out by Mme. Michel,

a nurse in Professor Pierre Marie's ward at the Salpêtrière.

Most of the diagrams were made by Mr Reignier.

The photographs and radiograms were taken under Mr Infroit's supervision.

Dr Henry Meige has been of invaluable assistance to us in looking after all the illustrations; and we desire to record here our great indebtedness to all our coadjutors.

CHAPTER II

THE MUSCULO-SPIRAL NERVE

The statistics of all the neurological centres prove that musculo-spiral paralysis is by far the commonest of peripheral palsies. The long course of the nerve which winds round the humerus in close apposition to the musculo-spiral groove accounts for this frequency, and, on the other hand, explains the fact that a fracture of the arm is usually complicated by musculo-spiral paralysis.

We must also draw attention to the vulnerability of the nerve, of which musculo-spiral paralysis from compression, or crutch paralysis, offers an example, and to its almost exclusively motor character.

At first sight it would appear that mixed nerves, all containing, as they do, motor and sensory fibres, vaso-motor and secretory filaments, ought, as a result of similar lesions, to present similar disturbances; but this is not so. Each mixed nerve has a very clearly defined clinical individuality; not only in the way it reacts to the lesion, but also in the manner in which it recovers its functions.[1]

ANATOMY

The musculo-spiral nerve arises from the brachial plexus in the axilla, where it is situated posterior to the median nerve and to the axillary artery. When it leaves the axilla it enters the musculo-spiral groove between the insertions of the internal and external

[1] Pierre Marie and Mme. Ath. Benisty, "Individualité clinique des nerfs périphériques," *Soc. de Neurologie*. March 15, 1915. *Revue neurologique*, May–June 1915.

heads of the triceps muscle, where it runs parallel to the superior profunda artery. Then it crosses the external intermuscular septum and appears on the anterior aspect of the arm in the outer bicipital groove at about 10 cm. above the epicondyle.

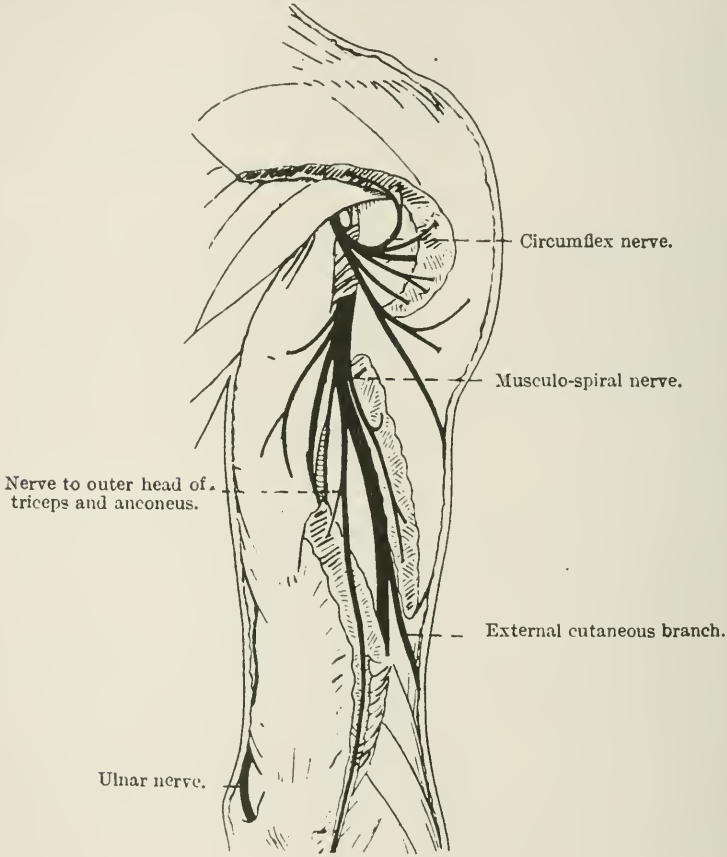


FIG. 4.—Musculo-spiral nerve.

At the level of, or a little below, the articulation, the nerve divides into two terminal branches.

The *anterior or sensory branch* lies first of all under the supinator longus, and then passes down the anterior aspect of the forearm. It supplies the outer half of the back of the hand (figs. 11 and 12).

The *posterior or motor branch*, the larger of the two, at about 2 cm. below the articulation penetrates the substance of the supinator

brevis to occupy the space between the two superficial and deep muscular layers on the posterior aspect of the forearm.

The first branch given off by the musculo-spiral nerve is the *internal cutaneous*. It starts where the nerve enters the musculo-spiral

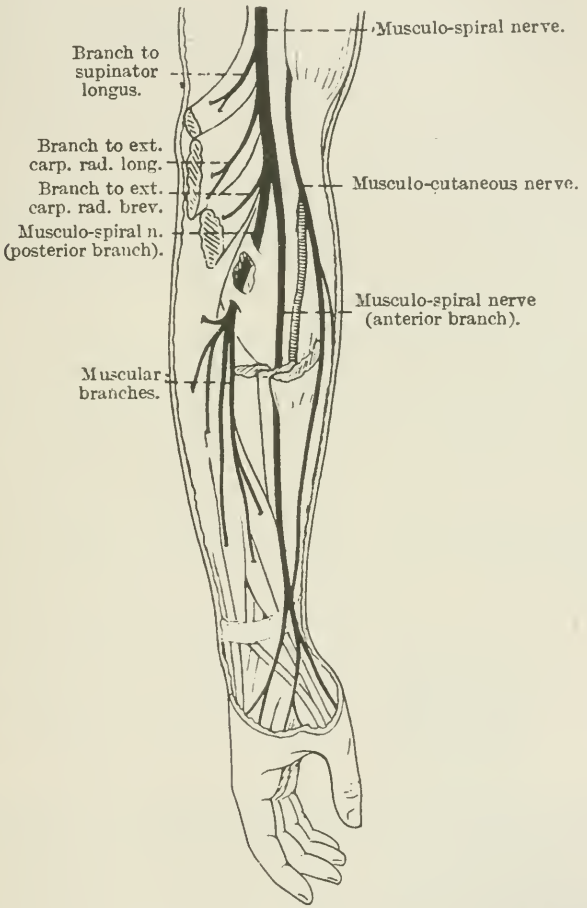


FIG. 5.—Branches of musculo-spiral nerve to forearm and hand.

groove; immediately beneath it is given off the *nerve to the long head of the triceps. The branches to the inner and outer heads of the triceps and to the anconeus muscle* spring from about the centre of this groove.

The *external cutaneous branch*, which supplies the posterior aspect

of the forearm (fig. 11), commences at the level of the lower third of the musculo-spiral groove before the nerve traverses the external intermuscular septum. This cutaneous branch perforates the outer head of the triceps and the brachial aponeurosis at about the level of the insertions of the supinator longus, and it supplies the skin.

The branch to the supinator longus is given off where the musculo-spiral nerve appears in the outer bicipital groove. *The branch to the extensor carpi radialis longior* arises a little below it. Before penetrating the supinator brevis and encircling the neck of the radius, the posterior branch gives a filament to the *extensor carpi radialis brevior.* In its passage under the supinator brevis it supplies it with two or three motor filaments.

All the remaining muscles of the posterior aspect of the forearm receive their branches from the posterior interosseous branch of the musculo-spiral nerve, which then divides into a number of secondary branches.

Pierre Marie, Henry Meige, and A. Gosset have recently investigated motor localisations of nerve-trunks by application of electricity to the nerves direct during surgical operations at La Salpêtrière. The results obtained enable us now to confirm the existence of a systematic distribution of the nerve fibres supplying the different muscles. In our second volume we shall deal with this more fully.

As regards the musculo-spiral nerve, these authorities have established the individuality of the nerve fibres intended respectively for the supinators, the extensors of the wrist, and the common and special extensors of the fingers.

It is in the antero-internal portion of the trunk of the musculo-spiral nerve, in the upper arm, that the fibres which supply the two radial extensors of the wrist are situated, whilst the fibres intended for the extensors of the fingers occupy the postero-internal portion: in the external portion the fibres of supination appear to lie.

NORMAL AND PATHOLOGICAL PHYSIOLOGY OF THE MUSCLES SUPPLIED BY THE MUSCULO-SPIRAL NERVE.

In considering the physiology of muscles the immortal works of Duchenne (of Boulogne) will be referred to. Duchenne, by local application of electricity to muscles, and by observation of cases of progressive atrophy in children and adults, has established the normal and pathological physiology of muscles. His statements have been confirmed in nearly every case.

Before describing the appearance of the hand in paralysis of the different nerves of the upper extremity, it is necessary to realise what is the natural position of the sound hand when at rest, the muscles being as nearly as possible completely relaxed and the forearm resting on a horizontal level (fig. 6).

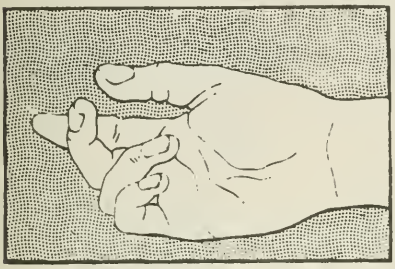


FIG. 6.—Position of *normal hand* in repose.

The position is then one of a slight degree of flexion and pronation of the wrist, which also inclines to the ulnar side. The fingers are flexed on the metacarpal bones; flexion of the phalanges is marked, especially the flexion of the second phalanx on the first, which occurs in all the fingers from the index to the little finger, and becomes more marked in each.

The thumb, besides being slightly flexed, shows pronounced opposition.

So that in the normal state the tone of the flexor and pronator muscles is superior to that of the extensors.

The musculo-spiral is the nerve of extension. In the upper arm it supplies the triceps (long, external, and internal heads) and the anconeus; in the forearm, the extensors of wrist and fingers (fig. 8).

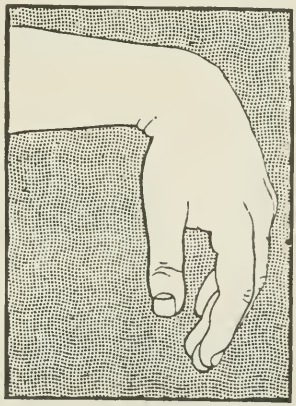


FIG. 7.—Attitude of hand in musculo-spiral paralysis (*wrist-drop*).

In musculo-spiral paralysis, therefore, there is immediate and very marked drop of the hand and fingers.[1] The wrist and the first phalanges

[1] Even at the time of injury it is quite common to see sudden contraction of the flexors of the fingers occur.

are flexed; the flexion is limp, without any contracture, and it is easily reducible (fig. 7).

MUSCLES.	NERVES.	ROOTS.
Sterno-cleido mast.	*Spinal accessory.*	(XI.) & C^3, C^3.
Trapezius.	,, ,,	C^3, C^4.
Deltoid.	*Circumflex.*	C^5, C^6.
Pectoralis major.	*Ext. anterior thoracic.*	C^5, C^6, C^7.
Infraspinatus.	*Suprascapular.*	C^5, C^6.
Teres minor.	*Circumflex.*	C^5.
Teres major.	*Nerve to teres major.*	C^5, C^6, C^7.
Pectoralis minor.	*Int. anterior thoracic.*	C^7, C^8, D^1.
Latissimus dorsi.	*Long subscapular.*	C^6, C^7, C^8.
Biceps.	*Musculo-cutaneous.*	C^5, C^6.
Brachialis anticus.	,, ,,	C^5, C^6.
Triceps.	*Musculo-spiral.*	C^6, C^7.
Supinator longus.	,, ,,	C^5, C^6.
Ext. car. rad. long.	,, ,,	C^6, C^7.
Anconeus.	,, ,,	C^7, C^8.
Ext. car. ulnaris.	,, ,,	C^7, C^8.
Ext. car. rad. brev.	,, ,,	C^6, C^7.
Ext. comm. dig.	,, ,,	C^6, C^7, C^8.
Abd. long. pollicis.	,, ,,	C^6, C^7.
Ext. brev. pollicis.	,, ,,	C^6, C^7.

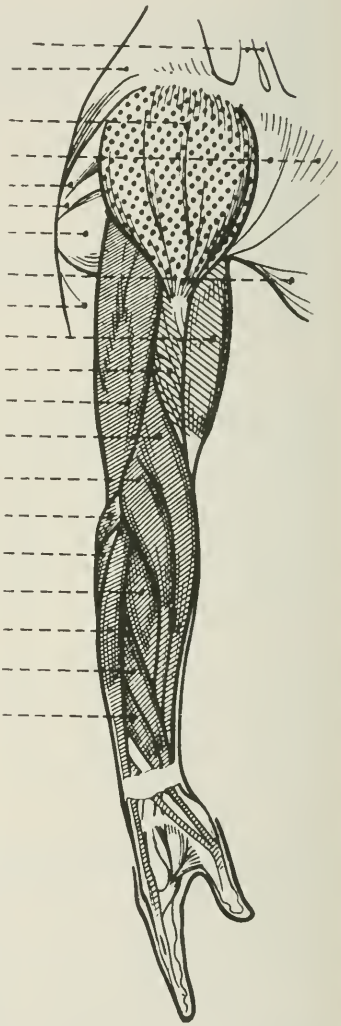


FIG. 8.—Muscles supplied by the *musculo-spiral nerve.*

Triceps. — The triceps forcibly extends the forearm on the arm. From Duchenne's experiments, it appears that the long head of the triceps is the weakest, on account of want of stability in its upper attachment; but either the external or internal head of the triceps can do all that is required, and when the triceps is entirely paralysed

the anconeus alone is capable of extending the arm.

When a wound of the posterior part of the arm has directly affected the nerve fibres of the external and internal triceps, or of all three heads, without injuring the trunk of the musculo-spiral nerve itself, this want of efficiency of the extensors of the forearm is not always easy to detect.

The patient can alternately flex and extend the forearm, holding a heavy weight in his hand; but if he is made to raise his elbow high enough, with his forearm flexed and his hand touching his shoulder, it becomes impossible for him to extend this bent forearm by reason of the weight alone.

Another way to test paralysis of the triceps is to extend the elbow and then to ask the patient to resist with vigour an attempt to flex the forearm on the arm.

When the lesion has involved the musculo-spiral nerve in the axillary region, the whole of the triceps is paralysed. As a result, then, of atrophy of the muscle and want of muscular power, passive flexion of the elbow may be more complete than in the normal condition.

Supinator longus.—The most common type of musculo-spiral paralysis is that consequent on injury to the middle third of the arm. This does not involve the triceps, but the supinator longus and all the muscles of the posterior part of the forearm are paralysed. Indeed, contrary to what occurs in case of injury to the median and ulnar nerves, partial, fascicular, or dissociated paralyses of the musculo-spiral are rare.

Involvement of the supinator longus is the result of a wound situated high up, but whatever the nerve lesion is in the upper arm, it is always accompanied by *total and general paralysis of all the muscles of the posterior part of the forearm.*

Is this due to weakness of the extensors, or, as has been maintained, to a greater length of their tendons ?

We think that the most probable explanation is the *essentially motor character of the musculo-spiral nerve.* Most of its fasciculi are motor. Its sensory fasciculi seem of minor importance, and only contain a few vasomotor, secretory, or other fibres.

The function of the supinator longus, in spite of its name, is to flex the forearm forcibly on the arm, the hand maintaining an intermediate attitude between supination and pronation.

In order to test whether the supinator longus is paralysed or not, the patient's hand should be pronated, and he should be asked to bend the forearm energetically. Whilst he does this, with one hand flexion should be opposed, and with the other the fleshy part of the muscle should be persistently palpated, the patient being told to bend and straighten his forearm alternately.

In case of musculo-spiral paralysis from a lesion above the branch to the supinator longus, flexion of the forearm is solely due to the action of the biceps and brachialis anticus. The prominence formed by the normal supinator longus at the bend of the elbow then disappears, and no muscular induration or tension can be found on most careful palpation.

Supinator brevis.—This muscle is a supinator exclusively; its contraction produces supination apart from any other movement.

In musculo-spiral paralysis the movement of supination is abolished. The supinator brevis is superseded during flexion of the ulna by the biceps—a flexor supinator *par excellence*; during extension of the ulna, by the external rotators of the shoulder, notably the infraspinatus; but these muscles can carry out only a weak movement of supination.

Extensor carpi radialis longior et brevior. Extensor carpi ulnaris.—The function of these three muscles is to extend the hand on the forearm.

The extensor carpi radialis brevior simply extends the hand.

The extensor carpi radialis longior extends and abducts the hand.

The extensor carpi ulnaris extends and adducts the hand.

The action of the extensor carpi radialis longior is more important than that of the extensor carpi ulnaris. When the patient forcibly extends his hand the three muscles contract simultaneously. In the case of a lesion of the musculo-spiral nerve, paralysis is almost always simultaneous in all three. It is only dissociated when the nerve lesion merely involves its posterior branch in the forearm below where the branches are given off to one or both radial extensors. We have also noticed this dissociation, though more rarely, in the case of incomplete musculo-spiral paralysis at the level of the brachial plexus.

To make the paralysis of the extensors of the hand apparent in musculo-spiral paralysis, care should be taken to keep the elbow and forearm of the patient motionless and resting on a table with the hand loosely suspended and pronated.

On palpation of the fleshy part of the three muscles at the upper and posterior part of the forearm, a little internal to the supinator longus and the external edge of the radius, no contraction is felt during attempts at extension ; whilst, on the contrary, there is energetic contraction of the flexors of hand and fingers. All that can be noticed is tension and dragging on the brachial aponeurosis, but the muscles themselves do not contract. Their tendons, which owing to the drooping position of the hand are very apparent at the level of the wrist, never move at all.

To verify abolition of *lateral movements* of the wrist, which are normally performed by the extensor carpi radialis longior and the extensor carpi ulnaris, possibilities of error arising from the movement of the elbow should be eliminated by resting the forearm on a table, as in examination for extension of the hand ; but the hand should be raised almost to the horizontal instead of being allowed to drop. Indeed,

Duchenne (of Boulogne) proved that, as the wrist bends, lateral movements diminish.[1]

Extensors of the Fingers (*Extensor communis digitorum, extensor indicis, extensor minimi digiti*).—The extensors of the fingers extend the first phalanx of the fingers on the metacarpal bones, and *only the first phalanx*. The extension of the second and third phalanges is performed by the interossei and the

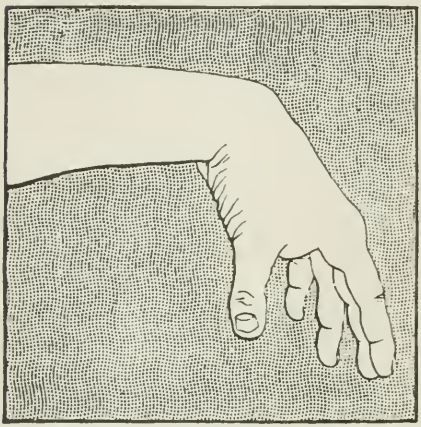


Fig. 9.—*Musculo-spiral paralysis.* Posture of hand in attempted extension of the fingers. Slight extension of last two phalanges can be carried out by the assistance of the interossei: but the first phalanx cannot be extended on the metacarpus.

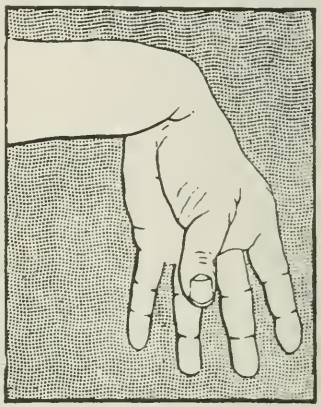


Fig. 10.—*Musculo-spiral paralysis.* Posture of hand and fingers in attempted abduction of the thumb.

lumbricales. The slight degree of extension of these two phalanges made by the patient in his efforts to extend the fingers on the metacarpus must not be attributed to restoration of the musculo-spiral nerve (fig. 9).

To demonstrate the immobility of the first phalanx, the patient's forearm should be rested on the table

[1] The action of the flexor carpi radialis as an abductor muscle, and that of the flexor carpi ulnaris as an adductor, is negligible. Duchenne denies that these muscles take part in any way in lateral movements of the wrist.

with the wrist semi-extended, and he should be asked to extend his fingers.

If the extensors of the fingers are paralysed, no movement of this kind will take place in the first phalanges.

Extensor longus pollicis. Extensor brevis pollicis. Abductor longus pollicis.—The extensor longus pollicis extends the two phalanges of the thumb, and simultaneously slightly extends the hand.

The extensor brevis pollicis is the only direct abductor muscle of the first metacarpal bone.

The abductor longus pollicis, a very powerful and long muscle, is inserted at the base of the first metacarpal bone of the thumb. Its function is to draw this bone obliquely outwards and forwards. In addition, its complete contraction flexes the hand and inclines it outwards, and during this time there is co-operative contraction of the extensor carpi ulnaris to keep the hand firm.

Abduction of the thumb is impossible in musculo-spiral paralysis, and all the patient's efforts only succeed in getting the thumb to the ulnar edge of the hand by use of the muscles in the thenar eminence. During these attempts the tendons of the anatomical snuffbox do not stand out in the slightest degree (fig. 10).

Accompanying Disorders of the Unaffected Muscles.—In musculo-spiral paralysis, some muscles, not supplied by the musculo-spiral nerve, and which have remained healthy, contract feebly and awkwardly.

The flexors, for instance, work badly on account of permanent flexion of the hand and the first phalanges. This posture, Duchenne says, will cause shortening of the flexors, and consequently constrained movement. The patient can neither close his fist nor flex all the phalanges completely; the third phalanges will flex badly or not at all; the thumb does not oppose the index fingers, the patient being obliged to turn it aside with his sound hand in order to put his four fingers into the palm of that hand.

The interossei muscles imperfectly carry out the

movements of bringing together and separating the fingers. The wrist-drop and paralysis of the extensors of the fingers impede these movements. They can be made with greater facility by placing the patient's hand flat on a table, the first phalanges being well extended.

Finally, extension of the thumb [1] and its opposition to the other fingers are defective.

These facts should be borne in mind to prevent attributing disorders, which are due to paralysis of the musculo-spiral nerve alone, to associated lesions of the median and ulnar nerves.

Electrical Reactions.—We have already pointed out that the musculo-spiral nerve, whatever may be the degree of the lesion with which it is affected, nearly always reacts by absolutely complete paralysis of the extensor muscles of the wrist and of the fingers, with the characteristic wrist-drop ; inability to extend the wrist and the first phalanges of the fingers ; and inability to extend and separate the thumb. Frequently this palsy is accompanied from the onset of the injury by complete reaction of degeneration, *i.e.* when the electrodes are applied to the most favourable motor points, faradic non-irritability of the nerve and muscles, galvanic non-irritability of the nerve, galvanic hypo-irritability more or less marked of the muscles, with slowness of the contraction, inversion, or equality of the polar formula and predominance of the longitudinal reaction, are noted.

Muscular Atrophy.—Muscular atrophy which is quite obvious from two to three months after the injury should be carefully noted, for it is most important in diagnosing the gravity of the lesion. When the nerve has been completely divided or badly crushed, atrophy of the muscle may be so marked seven or eight months after the injury that it looks

[1] Forcible contraction of the muscles of the thenar eminence may produce a slight extension of the second phalanx of the thumb, but this movement is never as complete or as strong as when the long extensor is spared.

as if the skin and the subcutaneous cellular tissue were adherent to the posterior aspect of the two bones of the forearm.

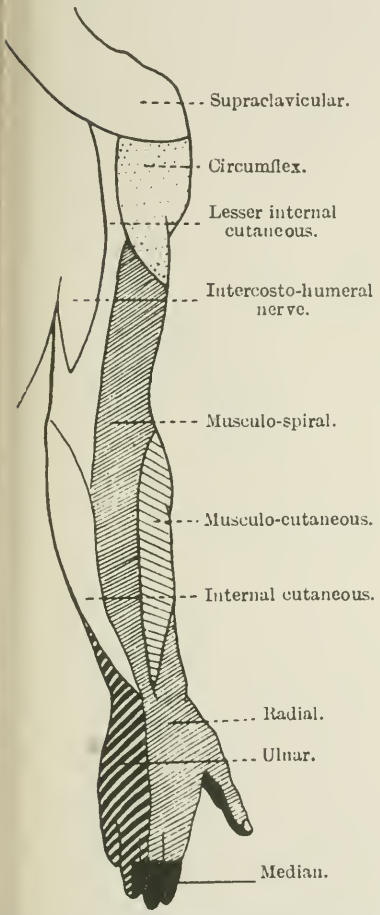


FIG. 11.—Peripheral sensory distribution of the upper limb (posterior aspect).

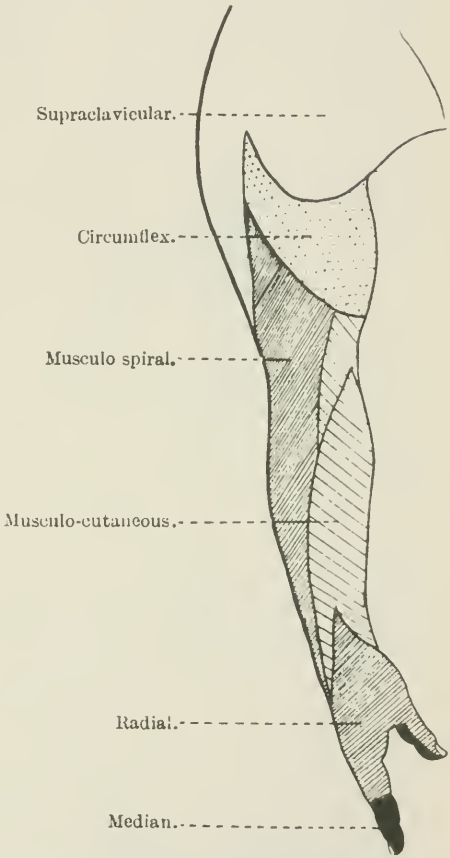


FIG. 12.—Peripheral sensory distribution of the upper limb (external aspect).

Sensory Disorders. — A. *Objective disturbances.*— Objective sensory disturbances following lesions of the musculo-spiral nerve demand most minute examination, for they are rather difficult to prove.

According to the text-book descriptions, the sensory area of the musculo-spiral nerve (figs. 11 and 12) compared with other nerves of the arm is very extensive, and yet, from the pathological point of view, in lesions of the musculo-spiral, sensory disturbances are least marked, even when the nerve has been completely divided.

In the commonest cases—lesion of the middle third of the arm—the internal cutaneous branch is rarely involved, whilst the external cutaneous is most frequently affected, and in every case lesion of the fibres which form the anterior branch of the musculo-spiral lower down should be complicated by marked sensory disorders within the region involved.

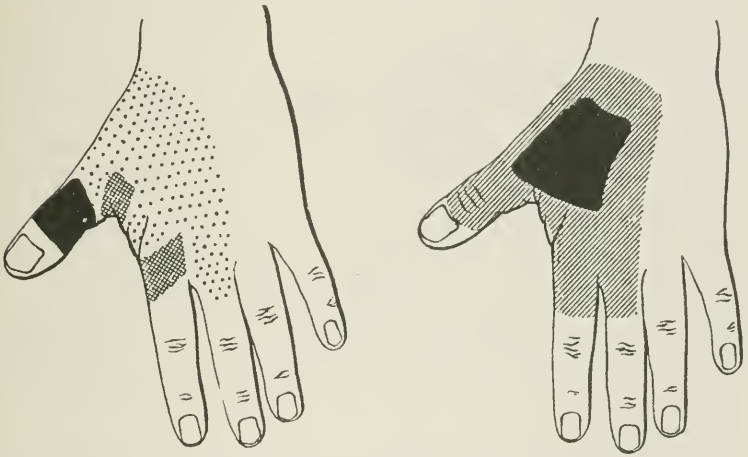
As a matter of fact, patients, of their own accord, only complain of slight numbness and loss of sensation in the first dorsal interosseous space and on the dorsal aspect of the thumb.

Much less frequently they draw attention to another part, more difficult to define accurately, which is situated on the upper and back part of the forearm.

In cases of lesion of the musculo-spiral nerve, sensibility should be most minutely tested and with extreme lightness of touch. Tactile disturbances should be tested with a wisp of cotton-wool, with the slightest possible pressure. These tactile disturbances will be found confined to the area indicated in figs. 13 and 14. Sensibility to heat and cold is impaired in the same area; frequently there is total thermal loss of sensibility, especially to heat. In some cases this thermal sensibility is only delayed. In testing, it may be noticed that if the patient hardly notices, or does not notice, heat and cold at all in the periphery of the zone of dysæsthesia, he is correct about his tactile sensations. Every time a tube is applied he feels the touch but not the temperature.

Sensibility to pin-prick, except over a very limited area, is almost unaffected; the patient feels each prick, but always asserts that it feels more like a touch than a prick.

To what is this discrimination of sensory disturbances in musculo-spiral paralysis due? We think that it is due to the fact that this nerve contains but a limited number of sensory fibres, and that these fibres only supply quite the superficial integument, so that a somewhat firm pressure acts on the deep sensibility, belonging to other nerves than the musculo-spiral. Belenki[1] thinks that this deep sensibility in



FIGS. 13 and 14.—Two frequent modes of distribution of sensory changes in complete division of the musculo-spiral nerve. *Black area*: complete anæsthesia, except to deep pricking, which often feels like a touch. *Grey area*: very marked hypæsthesia to pricking, very superficial anæsthesia to heat, cold, and touch. *Dotted area*: slight hypæsthesia to touch, heat and cold.

the area of the musculo-spiral nerve in the hand belongs to the median nerve.

No doubt, too, the assistance of adjacent nerves and the intercommunication of nerves which takes place in the deep layers as well as in the superficial tissue should be taken into account.

There is generally no disturbance in deep sensibility in musculo-spiral lesions. Baræsthesia, or perception of weight, remains unimpaired.

[1] Belenki, "Les symptômes sensitifs dans les sections anatomiques et physiologiques des nerfs périphériques," *Presse médicale*, Feb. 17, 1916.

Sensibility of bone is slightly diminished in the phalanges of the thumb ; this impairment of sensation is still slighter over the first metacarpal bone and the styloid process of the radius. Often sensibility at the level of the second and third metacarpal bones is not affected in the least, nor is that of the first phalanges of the index and middle fingers. The sense of position is generally well preserved, even in the case of the thumb.

B. *Subjective disturbances* are rare in cases of musculo-spiral paralysis.

The patient rarely suffers, except from pain consequent on fracture of the humerus, ankylosis of the elbow, and from adhesions. In 5 out of 100 cases, pricking, numbness, and neuralgic pain will be complained of in the region of the external cutaneous branch on the posterior aspect of the forearm and the dorsal aspect of the thumb. Damp weather, walking, and moving the injured limb aggravate these pains. They generally subside in a few months, though we have seen a case in which eighteen months after injury these symptoms persisted and were even exaggerated.

In about 1 in 100 cases of lesions of the musculo-spiral nerve, acute causalgia, similar to that described in the course of lesions of the median nerve, has been observed.

In one of these rare instances the burning pains were referred to the back of the hand, which was red and "puffy," to use Sicard's expression. There was a lesion of the upper part of the musculo-spiral nerve in the axilla, accompanied by involvement of the musculo-cutaneous.

Frequently, however, numerous painful spots at the level of the fracture of the humerus are noticed, and, when the nerve is not quite divided, over the head of the radius and all over the posterior aspect of the forearm along the course of the external cutaneous fibres. When the lesion is of moderate severity, and during the period of restoration of the nerve, these

painful spots are very frequent: they are much rarer—though they do occur—in the case of grave lesions, even when the nerve has been completely divided.

Tendon and Periosteal Reflexes should be examined by comparison with the sound side.

The *stylo-radial reflex* is sometimes completely abolished, and then the supinator longus not only does not contract (the lesion in that case being situated above the origin of its motor nerve), but the biceps itself does not contract at all; flexion of the fingers, more or less pronounced, is the only movement which occurs, with occasionally flexion of the wrist. The abolition of this reflex is most likely to be met with when the missile has considerably injured the fleshy part of the biceps. It is accompanied by a diminished sensibility of the bone at the level of the styloid process of the radius.

At other times this reflex is simply diminished. Sometimes it is as brisk as, or even brisker than, on the sound side; but in these latter cases the biceps only contracts, unless the supinator longus has escaped.

When the stylo-radial reflex has once been abolished, no matter how well restored from both motor and electrical points of view the long supinator has been, this restoration does not produce immediate reappearance of the reflex; its contraction on percussion of the styloid process of the radius is very slow.

The *ulnar pronator reflex* is sometimes exaggerated.

The *biceps reflex* shows more irregular changes. When the lesion is situated high up, the reflex is generally abolished and very rarely inverted. When there is a lesion of the middle third of the humerus it is sometimes brisk and sometimes normal, but more often diminished; especially when there is a cicatrix of any consequence on the body of the muscle, without interfering too much with its voluntary or electrical contraction.

The *triceps reflex* is just as liable to be affected as the tendo Achillis reflex.

EXPLANATION OF PLATE I.

A. *Musculo-spiral paralysis. The hand seen from the ulnar side is drooping, the wrist is flexed from paralysis of its extensors.*

There is also a distinct drop of the first phalanges of the fingers due to paralysis of their extensors.

The thumb inclines to the palm of the hand; the abductor longus and the extensors of the thumb being paralysed, the predominant tension of the thenar muscles draws it towards the ulnar side in a moderate degree of adduction and opposition.

B. *Musculo-spiral paralysis. Hand seen from the radial side.*

C. *One of the postures of the hand in a case of painful lesion of the median nerve.*

The fingers being extended and placed close together give the hand the appearance of the so-called "accoucheur's hand." In this case the flexors of the fingers are most paralysed.

D. *Associated lesion of median and ulnar nerves. Clawing of all the fingers, but most marked in the little, ring, and middle fingers.*

Considerable atrophy and flattening of the thenar and hypothenar eminences.

The thumb is on a level with the other fingers, the palm of the hand is widened and resembles the "ape-hand."

Atrophy of the adductor pollicis is seen; the flabby skin makes a large, soft fold in the space between the thumb and index finger.

E. *Eczematiform eruption, with large crusts covering the hand and fingers (both palmar and dorsal aspects) in a case of painful lesion of the median nerve.*

The patient for several months continuously made use of wet dressings and arm baths to relieve his pain.

A

B

C

D

E

PLATE I.

Idiomuscular Contraction is so liable to variations that we do not think useful information on the nature of the lesion can be deduced from it.

In the early stages of injury idiomuscular contraction is stronger; especially if there has been fracture of the bone. Later on, it diminishes in intensity, the contraction is slower, and the diminution is in relation to the atrophy of the muscle. In grave lesions, idiomuscular contraction entirely disappears when there is considerable atrophy.

Idiomuscular contraction of the triceps which is rarely paralysed is nearly always strong; but it may also be normal or even diminished.

Vasomotor and Secretory Phenomena.—Disturbances of this kind are absent or very slight, and only become important when there is affection of some other nerve or considerable vascular, osseous, articular, or muscular mischief.

When the hand is swollen, purple, and succulent in appearance, inquiry should be made whether the patient does not always hold it hanging down, or if the apparatus or splint he is wearing to raise his hand is not unduly constricting the return of the circulation.

The affected hand should be compared with the sound hand to eliminate the possibility of congenital acrocyanosis, which is of frequent occurrence and in which the symptoms are always bilateral.

However, apart from any constriction, the hand sometimes appears œdematous. This œdema is transitory and probably due to inactivity of the muscles on the posterior aspect of the forearm, contraction of which normally aids in the return of the circulation. This inactivity would also account for the induration of the skin on the posterior aspect of the forearm, which can be easily noticed by pinching the skin of that region between two fingers in comparison with the sound side.

Perspiration is very little affected by a lesion of the musculo-spiral nerve. We have sometimes come across excessive perspiration of the palm of the hand

and in the axilla on the side affected; but this was nearly always associated with a fractured humerus, much damage to the bone, and prolonged suppuration. In such cases it is not easy to point to the exact cause of the phenomena.

Trophic Disturbances only occur in association with a vascular lesion, and are not seen in ordinary cases. Desquamation of the epidermis, however, may sometimes be noticed at the level of the first dorsal interosseous space.

The relative atrophy of the paralysed hand is mainly due to the immobility of the limb, which causes a diminution in size of the entire hand, and to the inward retraction of the thumb towards the palm, which causes the hand to shrink. Decalcification is very slight in musculo-spiral paralysis when there is no accompanying destruction of the bones or suppuration of long standing.

Tumour of the wrist is an everyday occurrence in musculo-spiral paralysis, and bears no relation to the gravity of the lesion. It is due to distension of the ligaments of the wrist, which makes the second row of bones protrude; it is also due to thickening of the synovial membranes, of the same nature as the subcutaneous infiltration already mentioned surrounding the tendons of the extensors.

The more or less pronounced wrist-drop, characteristic of musculo-spiral paralysis, depends on the degree of muscular atrophy and the distension of the ligaments of the carpus, as well as on the tone of the muscles, which usually varies greatly with the patients.

RECOVERY OF POWER OF MOVEMENT.[1]

The radial muscles are the first to begin to contract in the event of recovery of the musculo-spiral nerve.

[1] In the paragraph dealing with recovery of motor power and each nerve affected, we shall deal with the recovery of motor power only; recovery of sensibility and electrical contractibility will be specially dealt with in the second volume entitled *Treatment and Restoration of Nerve Lesions*.

In judging of the amount of return of voluntary contraction, causes of error arising from the assistance given by other muscles, and the patient's own endeavours to use his hand, should be eliminated.

Though considerably limited, the use of the hand in musculo-spiral paralysis is still possible. The patient can dress himself with his affected arm ; often he can hold a fork to eat with, or even write if clever and energetic. Some patients, when trying to write, support the affected right hand with the left and push it along the paper. They write slowly and the writing is small. Others again, keep the right hand still and move the paper with the left as they write.

By gradually bringing into play all the accessory muscles they get unforeseen assistance.

To judge of the extent of recovery in the power of movement, the paralysed forearm should be held quite still on a table and the extensor muscles of the wrist and fingers should be carefully palpated whilst prescribed movements of extension are performed. It should also be borne in mind, as Duchenne (of Boulogne) says, that voluntary extension of the fingers on the metacarpal bones is normally accompanied by co-operative and involuntary contraction of the flexors of the hand ; so that in the course of musculo-spiral paralysis energetic contraction of the flexors of the wrist simulates a slight elevation of the wrist and is a cause of error to be avoided.

Another reason for *careful palpation of the muscles of the extensors and flexors during attempted extension* is, that when extension of the wrist is so slight as to be doubtful, or possibly due to the patient's own manœuvres, palpation of the muscles aids in prognosis : if the muscles are felt to harden during the attempt at extension, this is a sign that movement will shortly be restored.

The examiner must, however, satisfy himself that the contraction noted is actually in the muscles and not due to mere tension of the aponeurosis of the forearm by the epitrochlear muscles.

Further, he should make sure that it is the radial or extensor muscles which are recovering their power, by telling the patient to raise his hand several times over, and during this attempt palpating the tendons at the level of the wrist.

To find out if restoration of movement is impending, place the patient's elbow on the table with the forearm raised perpendicularly, and the hand extended; then ask the patient to keep it extended and not to let it drop. If the muscles are beginning to regain their tone, the patient will be able to keep the hand in position for a few seconds, and in any case the drop of the hand will not be as sudden as in the early stages of the disease.

The following are some of the signs of impending recovery of power of movement in musculo-spiral paralysis described by Prof. Pitres in his report to the Neurological Society:[1]—

1. Gradual disappearance of *automatic pronation.*

The forearm in musculo-spiral paralysis is kept in pronation; if it is placed in supination and then released, it promptly resumes its normal position as if it were moved by a spring. When recovery of movement begins, the return to pronation is not so sudden.

2. Diminution in the sudden *flail-like drop of the hand.* When the forearm is placed in a perpendicular position with the elbow on a level, if it is alternately extended and flexed with the hand passive, the fall of the hand in extension is more gradual and moderate, as it is held up by the tone of the flexors: when it occurs in flexion, it is more sudden and complete, as the tone of the extensors has been abolished. Diminution in the suddenness of the drop of the hand in flexion shows return of tone to the extensor muscles.

3. *Diminution of the elastic flexion of the fingers.* If you lay the back of the hand of a patient suffering

[1] "The Value of Clinical Signs in Injury to the Peripheral Nerves, assisting in recognition of (A) lesion of the whole nerve; (B) the recovery of its functions," Report to the Neurological Society at the meeting on 6th April 1916, *Revue neurol.*, April–May 1916, pp. 486–7.

from musculo-spiral paralysis on the table, the fingers remain semi-flexed ; but if, after having completely extended them, you leave them alone, they promptly return to the position of semi-flexion. Diminution of this automatic flexion is a sign that the extensors are regaining tone.

4. The transfer of *voluntary motor impulses* to the antagonists of the paralysed muscles. Pitres says that this phenomenon is noticed for a few weeks before voluntary contraction returns. When the patient tries to lift his hand, the wrist does not move ; but the fingers of the hand stiffen and there is excess of flexion of their first phalanges, with the result that the fingers are shaped like a spiral staircase, of which the lowest rung would be represented by the little and the highest by the index-finger.

Dr and Mme. Dejerine[1] call this last sign in question, and regard this contraction merely as the normally predominant action of the interossei and flexors.

5. In addition, patients suffering from musculo-spiral paralysis, during the early stages of recovery, can extend the wrist when it is placed in semi-supination ; but if it is in complete pronation, the greater opposition to movement prevents straightening.

The *order in which movement is restored* to the muscles is nearly always the same.[2]

The common extensor is only restored after the radial muscles. The middle, ring, and little finger begin to extend first, and lastly the index ; but sometimes it is the index and middle fingers which first quite recover their movement ; the extensors of the thumb and the abductor longus regain their power much more slowly and last of all.

The supinator longus generally recovers its movements before the abductor longus pollicis. Whilst restoration is taking place, and during attempts at

[1] *Société de Neurologie*, April 6, 1916.

[2] Pierre Marie and Mme. Ath. Benisty, " Individualité clinique des nerfs périphériques," *loc. cit.*

extension of the wrist, frequently the supinator longus contracts vigorously. This is probably due to a kind of error in the selection of the point of entry of the nerve influx (A. Thomas). The branches given off highest up are not the first to recover, as might logically be supposed, from the shorter distance that the axis-cylinders would have to traverse in the course of repair.

In the great majority of cases the extensors recover their functions before the supinator longus. More rarely, it recovers its movements first, or just after the radial muscles.

Signs of Recovery.—*When can musculo-spiral paralysis be said to be cured?* As the amount of movement recovered is defective for months, this question is one that should be asked. It is a long time before the patient can raise his wrist with any vigour without at the same time flexing his fingers to some extent. *Simultaneous extension of the wrist and all the fingers seems to be the final stage in the recovery of voluntary movement.* This applies also to the separate extension of each finger. Prof. Pitres has suggested the two following tests to find out whether recovery from musculo-spiral paralysis is complete :—

Ask the patient to put his arm in the *attitude of a man taking an oath*, with wrist and fingers extended horizontally, the thumb raised and separated from the first finger, and with the tendons of the extensors and of the long abductor of the thumb marking out the anatomical snuffbox ; then ask him to supinate his hand.

The second test is simpler still. With the patient standing in front of him, the doctor should tell him to *place his little finger on the seam of his trousers* with his fingers well extended, and with the palm of his hand turned to the front. His musculo-spiral paralysis is cured if he can do this correctly.

The strength of the restored muscles returns very gradually long after complete recovery of voluntary movement : it can be measured by the dynamometer.

DIAGNOSIS

Diagnosis of musculo-spiral paralysis would be simple enough if it could not be simulated by various conditions due to war injuries.

The most curious condition is that lack of power in the hand described by Henry Meige as "congealed hand." The wrist-drop, though suggestive of musculo-spiral paralysis, is neither complete nor limp. Electrical reactions are normal or slightly modified in this condition (fig. 15).

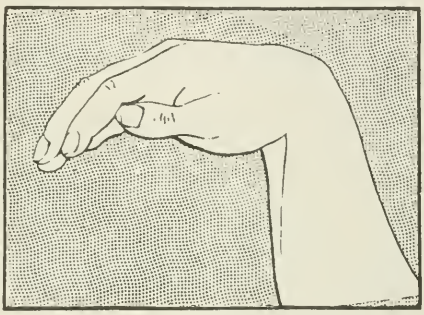


FIG. 15.—*Congealed hand.* (H. Meige.)

More or less marked drop of the hand can also be seen following direct muscular lesion of the radial muscles without paralysis of the nerve. Extensive adhesions may also impede the action of the extensor muscles.

Finally, some vascular lesions of the tendons or bones of the forearm cause marked ankylosis of the wrist, which simulates musculo-spiral paralysis.

In painful lesions of the median nerve (see p. 69) of some months' standing, the patient in his desire to relax the nerve-trunk and protect the skin of the palm of the hand, which becomes very painful, puts himself into attitudes of defence, which most commonly result in ankylosed flexion of the elbow and wrist. This flexion rather suggests musculo-spiral paralysis.

A correct diagnosis, in the various conditions described, can, however, be formed after clinical and electrical examination.

Moreover, some tests have been suggested in a monthly report to the Health Department by Professors Pitres, Testut, and Carrière for dispensing with

electrical examination in diagnosing musculo-spiral paralysis.

The tests are briefly as follows :—

1. *Clasped hands.* In musculo-spiral paralysis, when the patient clasps his hands as in prayer, he can usually resist any attempt to unclasp them, as the flexors lock them.

In the condition described as "congealed hand," the various movements required to carry out this test cannot be made, because the fingers are not limply bent but rigid.

2. *Clapper test.* In making this test, the patient claps the palms of his hands together like a clapper, his wrists representing the hinge of the clapper. Whilst he is keeping his wrists firmly fixed together, he should be told to separate his fingers : if they are paralysed, they will bend at once ; if the wrists are suddenly separated afterwards, the one on the paralysed side will immediately become flexed and pronated.

In the condition known as "congealed hand," or in ankylosis of the wrist, the affected hand cannot be placed quite close to the sound hand ; moreover, if the two hands are separated, the fingers remain fixed and rigid, for the wrist only drops very slightly after the passive straightening it has undergone.

3. *Laying the hand flat on the table.* A patient with musculo-spiral paralysis can lay the palm of his hand flat on the table, but not the back of it, as the fingers remain slightly bent.

In the "congealed hand" or ankylosis the patient can neither do one nor the other.

4. *The clenched fist* sign, which we have already referred to above.

5. *Extension of the thumb in abduction* cannot be performed in musculo-spiral paralysis. The "congealed hand" can sometimes make the movement in spite of very frequent retraction of the thumb in adduction.

Finally, we should like to urge the importance of

most careful clinical and electrical examination, taking each lesion into careful consideration in the course of the examination, and not being content with a superficial diagnosis, in cases of musculo-spiral paralysis complicated by lesions of bones, tendons, and joints.

CHAPTER III

THE MEDIAN NERVE

Contrary to what is observed in lesions of the musculo-spiral nerve, which are manifested by a form of paralysis which is almost always identical in character, lesions of the median nerve give rise to various syndromes which may be divided into two classes —*painful* and *painless lesions*. The latter should be subdivided into *total* and *partial*, or dissociated, paralysis, for it frequently happens that slight injuries may cause a lesion of some of the fibres only, without involving the nerve-trunk at all.

As we have already mentioned, it has been definitely proved that the branches intended for the different muscles are quite distinct long before they quit the nerve-trunk.[1]

ANATOMY

The anatomy of the median nerve will assist in explaining its pathology. This nerve is unquestionably the most important nerve of the upper extremity; its distinctive functions are sensory, vasomotor, and, as it were, trophic, as well as motor. The only other nerve that possesses these functions to the same extent is the sciatic.

The muscles supplied by the median nerve are the pronator radii teres, flexor carpi radialis, palmaris longus, flexor sublimis digitorum, flexor longus pollicis, flexor profundus digitorum, the lumbricales of the second and third fingers, the pronator quadratus, and, lastly, most of the muscles in the thenar eminence (opponens, abductor brevis, and the outer head of the flexor brevis pollicis). Briefly,

[1] P. Marie, H. Meige and Gosset, "Les localisations motrices dans les nerfs périphériques," *Académie de Médecine*, Dec. 28, 1915.

the median nerve is the nerve of pronation, flexion of the wrist and fingers, and opposition of thumb.

The two roots of the median nerve embrace the lower part of the axillary artery: in the arm this nerve lies at first on the outer side of the brachial artery, which separates it from the ulnar nerve, the internal cutaneous and its accessory.

At first, pressed close to the coraco-brachialis, the median nerve

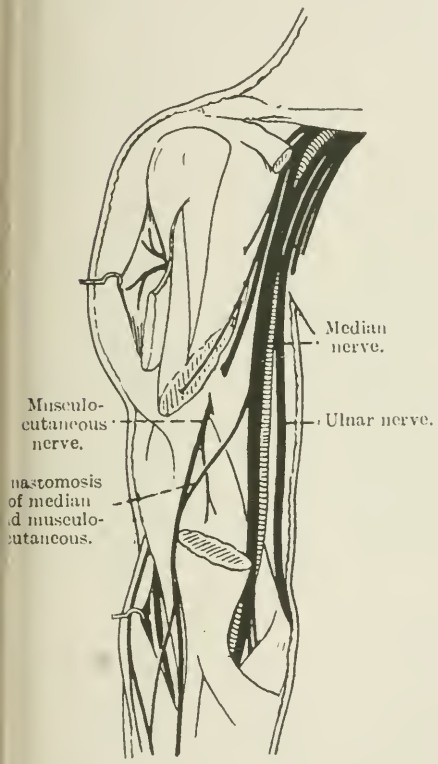


FIG. 16.—Nerves of the arm.

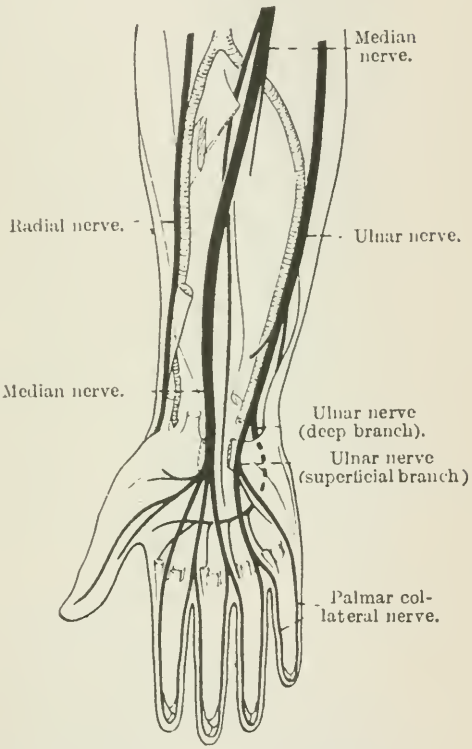


FIG. 17.—Nerves of the forearm and hand.

in the arm follows the internal border of the biceps, which sets it back against the brachialis anticus. There are no muscles on its inner side, especially in persons whose muscles are not well developed. This inner side corresponds to the brachial aponeurosis and skin.

At the bend of the elbow the median nerve leaves the brachial artery and passes between the two heads of the pronator radii teres. In the forearm this nerve really earns its name and descends in

the middle of the anterior aspect of the arm between the flexor sublimis and the flexor profundus digitorum.

With the exception of the *upper nerve to the pronator radii teres*, and a few twigs to the elbow-joint which all arise above the internal condyle of the humerus, the median nerve gives off no branches in the arm. It is in the forearm, about 2 cm. below the bend of the elbow, that the median supplies its principal muscular branches. The anterior branches supply the pronator radii teres, flexor carpi radialis, palmaris longus, and the flexor sublimis digitorum. The posterior branches supply the flexor longus pollicis and the two external fasciculi of the flexor profundus digitorum. The *anterior interosseous nerve* arises at this level and accompanies the anterior interosseous artery along the interosseous membrane: it supplies the *pronator quadratus* and is lost in the soft parts of the wrist-joint.

The *palmar cutaneous branch* arises from the median nerve at the lower part of the forearm just above the wrist; it supplies the thenar eminence and outer part of the palm of the hand.

Finally, at the level of the thenar eminence, a little internal to its inner border, the nerve divides into six terminal branches. One is a muscular branch which supplies the *abductor, opponens,* and the *outer head of the flexor brevis pollicis.* The others send motor branches to the *first and second lumbricales*, and supply the skin of the thumb, index and middle fingers, as Plate III. (p. 92) shows.

Anastomosis takes place between the median nerve and most of the nerves of the arm. Two such anastomoses are of frequent occurrence—one with the musculo-cutaneous, in the arm; and the other, in the palm of the hand, with the ulnar nerve. These anastomoses should be borne in mind, as they may serve to explain certain anomalies observed in the course of lesions of the median nerve.

PHYSIOLOGY

The normal physiology of the muscles of the forearm and hand may with advantage be reviewed in order to thoroughly understand the functional disturbances of the muscles that complete section of the median nerve may cause (fig. 18).

The *pronator radii teres* pronates and flexes the forearm, and the *pronator quadratus* is also a strong pronator of the forearm.

Flexion of the hand on the forearm is carried out by (1) *the flexor carpi radialis,* which flexes the outer portion of the hand without imparting lateral movement to it; (2) the *palmaris longus,* which flexes the whole of it; (3) the *extensor carpi ulnaris,* which flexes the fifth metacarpal more; (4) the *abductor longus pollicis,* though not nearly to the same extent.

Flexion of the hand generally is effected by the co-operative action of these different muscles. In addition, the flexors of the

hand contract sharply but involuntarily, when there is voluntary extension of the fingers on the metacarpus.

Whatever may be said to the contrary, these muscles, even

MUSCLES.	NERVES.	ROOTS.
Deltoid.	*Circumflex.*	C^5, C^6.
Pectoralis major.	,,	C^5, C^6, C^7.
Triceps.	*Musculo-spiral.*	C^6, C^7.
Biceps.	*Musculo-cutaneous.*	C^5, C^6.
Brachial. anticus.	,, ,,	C^5, C^6.
Pron. radii teres.	*Median.*	C^6, C^7.
Supinator longus.	*Musculo-spiral.*	C^5, C^6.
Ext. carpi radialis (long. and brev.).	,, ,,	C^6, C^7.
Flexor carpi rad.	*Median.*	C^7, C^8, D^1.
Palmaris longus.	,,	C^8, D^1.
Flexor sub. dig.	,,	C^8, D^1.
Flexor carpi ulnaris.	*Ulnar.*	C^8, D^1.
Thenar muscles.	*Median.*	C^6, C^7.
Hypoth. muscles.	*Ulnar.*	C^8, D^1.

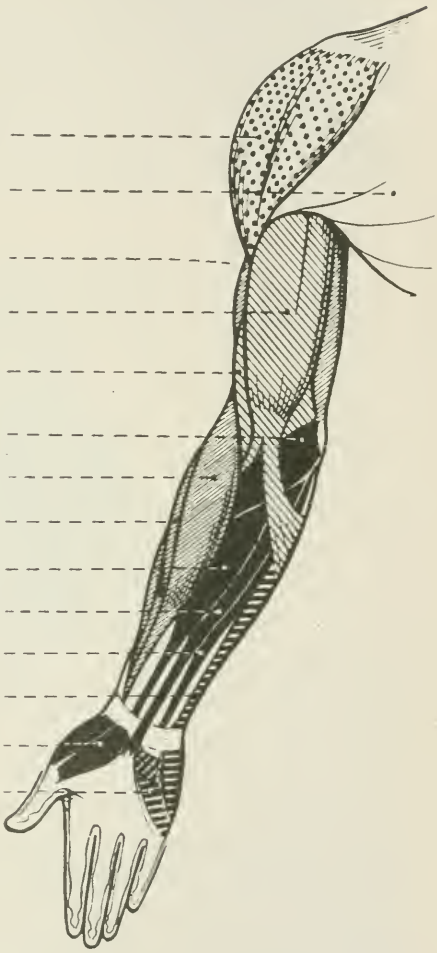


FIG. 18.—Innervation of the muscles of the upper extremity (anterior aspect).

including the flexor carpi radialis, give no lateral movements to the hand.

The *flexor sublimis digitorum* flexes the second phalanx on the first, the third phalanx remaining extended: the *flexor profundus digitorum* flexes the second and third phalanges on the first. Their action on the first is very weak or *nil*.

The synergic action of the extensors of the hand and fingers

increases the power of the flexors. Complete flexion of the fingers, as in the action of clutching, is only possible when the extensors are contracted to the fullest extent.

The *flexor longus pollicis* only flexes the last phalanx of the thumb.

The *first and second lumbricales* act like the interossei into which they are inserted; they cause flexion of the first phalanges and extension of the second and third.

The lumbricales do not assist in lateral movement, with the exception of the first, which abducts the first phalanx of the index finger slightly.

The *muscles of the thenar eminence* have been divided by Duchenne

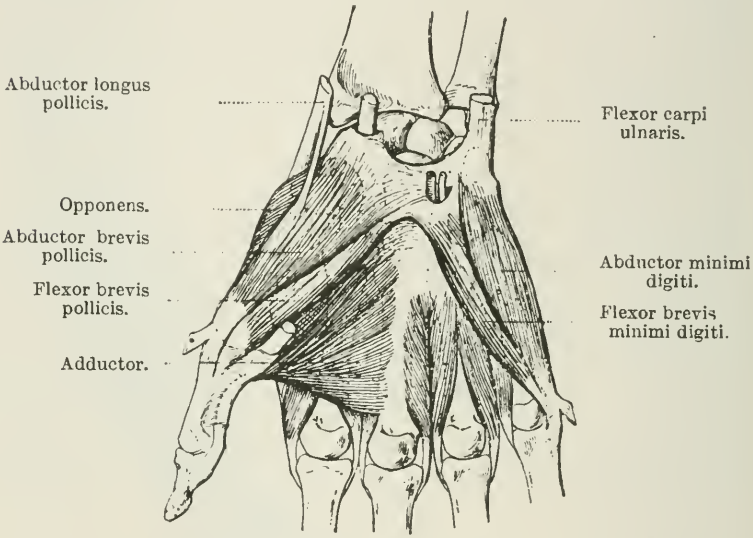


FIG. 19.—Muscles of the hand.

(of Boulogne) into two groups: (1) Those which are inserted into the outer side of the first phalanx of the thumb (abductor pollicis and the outer head of the flexor brevis pollicis); and (2) those which are inserted into the internal sesamoid on the inner side of the first phalanx of the thumb (adductor transversus and inner head of the flexor brevis pollicis) (fig. 19).

The *opponens pollicis* should be considered separately. It flexes the first metacarpal sharply on to the wrist and draws it inward at the same time.

The *abductor pollicis and the outer head of flexor brevis pollicis* give the first metacarpal a movement similar to that made by the opponens pollicis; but they simultaneously extend the second

phalanx and bend the first on its outer side, making it revolve from without inwards on its axis, so that the tip of the thumb is opposed to the tips of the first two fingers.

The outer head of the flexor brevis pollicis bends the first metacarpal and the first phalanx of the thumb towards the little finger more than the abductor does: it aids in opposition to the ring and little fingers. It is not, however, so useful as the abductor brevis, which flexes the first metacarpal; anyone having lost the use of the abductor would be obliged to make "a claw hand" in order to oppose the thumb to each of the fingers, *i.e.* to extend the first phalanges and then to bend the last two sharply.

Clinical Varieties of Lesions of the Median Nerve

We will study the lesions of the median nerve in the following order :—

I. Painless lesions.
   A. Total paralysis.
   B. Partial paralysis.
II. Painful lesions—Causalgia.

I. PAINLESS LESIONS

A. *Total Paralysis*

Appearance of the Hand.—When the median nerve has been completely divided, or when there is a grave lesion of this nerve due to wounds situated on the inner aspect of the arm, the patient's affected hand is seen to be larger than the sound one, owing to the influence of the adductor pollicis, which puts the thumb on a level with the four fingers and produces the so-called "ape-hand."

In addition, the whole of the hand inclines slightly to the ulnar side of the arm (figs. 20 and 21). The index and the middle fingers, without being completely extended, are more extended than normally. There is considerable atrophy of the muscles of the thenar eminence, particularly of its outer part.

Motor Disorders.—After considering the play of the joints and the density of the adhesions from scars, the

EXPLANATION OF PLATE II.

A. *Ulnar paralysis of the right hand from lesion of the nerve above the internal condyle of the humerus. The left sound hand is for comparison.*

Note complete absence of clawing;

Great wasting of the first dorsal interosseous spaces;

Less wasting of the last three interosseous spaces;

Impossibility of separating the fingers;

A slight degree of abduction of the little finger from paralysis of the hypothenar muscles.

B. *Total paralysis of the median and ulnar nerves on the right side from division of the two nerves at the arm. Severe concomitant lesion of the brachial artery.*

Comparison with the left sound hand shows an "ape-hand" appearance.

Considerable atrophy of the thenar and hypothenar muscles.

Permanent abduction of the fingers, and a succulent and œdematous appearance of their terminal phalanges.

Though there is complete paralysis of the flexors of the fingers and interossei, the phalanges of fingers and thumb are in slight flexion.

C. *Ulnar paralysis, following paralysis of all the nerves of the arm, due to a reduced dislocation of the shoulder.*

The musculo-cutaneous and musculo-spiral nerves recovered uneventfully; but there was pain in the palm of the hand and in the fingers, with clawing of all the fingers, before the median and ulnar nerves recovered.

The profile of the hand shows flexion of the fingers and much atrophy of the first dorsal interosseous space.

A

B

C

PLATE II.

degree of movement will be dealt with. It is best to begin with the shoulder and to study the movements of each of the muscles of the arm in turn, no matter what the site of the lesion.

In cases of a pure lesion of the median nerve, flexion and extension of the elbow are not affected, and supination is also unimpaired.

Paralysis of the pronators.—Pronation is incomplete

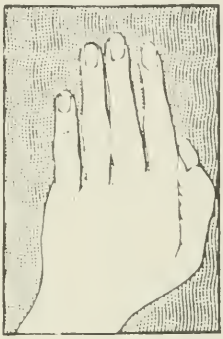


FIG. 20.—*Paralysis of the median nerve.* "Ape-hand" (dorsal aspect).

FIG. 21.—*Paralysis of the median nerve.* "Ape-hand" seen from palmar surface.

and defective. When the forearm is flexed, the patient supplements this defect by holding his elbow out; when it is extended, he rotates the arm inward, with passive pronation of the hand as a result. In the first case the deltoid assists in pronation, and in the second case the subscapularis comes into play. By attempted extension of the hand and fingers, especially of the extensor carpi ulnaris, and perhaps also of the extensor communis, and some apparent contraction of the long

supinator, the patient at the same time succeeds in pronating his hand.

Paralysis of the palmar muscles.—When the patient is told to flex his wrist, and when, by careful palpation of the tendons at the bend of the wrist, this flexion is opposed, it can be seen that the tendon of the flexor carpi ulnaris only is much stretched; during this movement the hand is drawn towards the ulna. The palmar tendons remain flaccid; and, even when at rest, the tendons of the palm of the hand on the sound side are more prominent than in the affected hand.

Paralysis of the flexor sublimis and outer fasciculi of the flexor profundus digitorum.—Flexion of the ring and little fingers is fairly good, though weaker than normal, on account of the action of the flexor profundus, which flexes the third phalanx and thereby involves the second.

There is absence of flexion in the index finger, and the middle finger only makes a feeble attempt at this movement; this finger is usually influenced by the movement of the ring finger, and its deep flexor may often be supplied by the ulnar nerve.

If a patient whose median nerve is injured is asked to show his fist, the index and middle fingers will be seen to have their phalanges slightly flexed. This purely passive movement is due to a kind of influence exerted by the fourth and fifth fingers. If this influence is counteracted by asking the patient to flex the index and middle fingers only, he cannot flex the index at all, and makes only a slight movement with the middle finger. This fact can be plainly demonstrated if the forearm is prolonged by the hand being held out straight with the phalanges of the second and third fingers motionless.

It should be remembered, too, that, in spite of complete paralysis of the flexor sublimis and the external fasciculi of the flexor profundus, when at rest the second and terminal phalanges of these fingers are not over-extended. Some tonus seems to persist in the

flexors, for there is a very slight tendency to flexion of the joint of the first and second phalanges.

Adequate flexion of the first phalanx of the fingers on the metacarpus is generally seen, and sometimes flexion is perfect in all the fingers, including the index.

Flexion of the first phalanx of the fingers, with extension of the last two phalanges, is carried out very well by means of the interossei in spite of paralysis of the first and second lumbricales.

Paralysis of the flexor longus pollicis.—Flexion of the second phalanx of the thumb is impossible, as the flexor longus pollicis is paralysed. This can be readily seen by first immobilising the first phalanx.

Paralysis of the thenar muscles.—Opposition and abduction of the thumb are very defective. The patient sometimes uses his adductor instead ; at other times he uses the inner head of the flexor brevis. By means of his adductor he is able to bring his first metacarpal into proximity with the second ; then the thumb will endeavour to creep along the first phalanges of the fingers, but without definitely extending its second phalanx, and without opposing its tips to the other fingers. It makes only a faint movement of opposition with its inner border, and this opposition is ineffectual, as the second and third fingers cannot be flexed, and the thumb does not incline inward enough to reach the tips of the fourth and fifth fingers.

Amyotrophy.—From the earliest stage of the injury marked flattening in the outer part of the thenar eminence may be noticed. If the lesion is of some months' standing the group of muscles arising from the internal condyle is also considerably atrophied ; in well-developed subjects especially the thinness of these muscles is in sharp contrast with the prominence of the supinator longus. The skin is colder at the level of the atrophied muscles.

Electrical Changes.—Reaction of degeneration occurs in all the paralysed muscles. As time goes on and atrophy increases, the lessened galvanic irritability

of the muscles is accentuated to such an extent that in a few months' time the nerve and the muscles

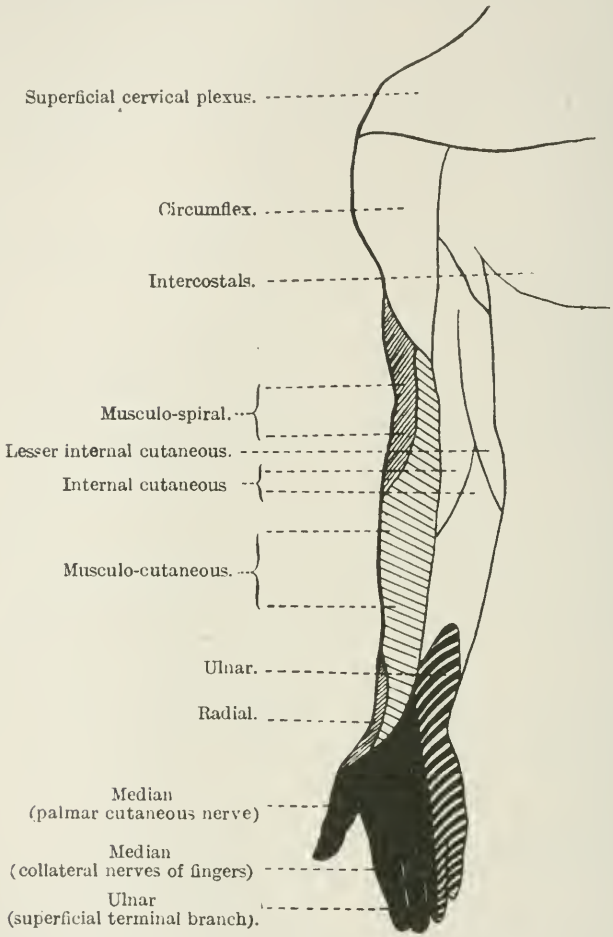


FIG. 22.—Peripheral sensory distribution of the upper limb (anterior aspect).

are insensible to either th interrupted or constant current.

Sensory Disorders.—The sensory changes form an interesting study.

The sensory distribution of the median nerve,

as shown in text-books (figs. 11 and 22), is altered to some extent.

Sensibility over a large area of the skin of the thenar eminence and over the outer part of the palm of the hand may hardly be affected at all in a severe lesion of the median nerve.

Frequently, too, the palmar aspect of the first phalanx of the thumb and now and then the tip of

Palm of the hand.

FIG. 23.

Back of the hand.

FIG. 24.

Distribution of sensory changes in severe lesions of the median nerve. *Black*. complete anæsthesia to all modes of sensation. *Grey*: hypæsthesia to pricking, anæsthesia to heat and cold. *Dotted portion*: less marked hypæsthesia.

the thumb are not completely insensitive to stimulation by heat or pain (figs. 23 and 24).

There is, however, nearly always complete anæsthesia in the index finger on both aspects, principally in the last two phalanges, and this also applies to the same phalanges of the middle finger.

Slight touch, pressure, pricking, heat and cold, are not felt. In the index finger the sense of position is usually abolished. There is complete anæsthesia

to vibrations of the tuning-fork in the last two phalanges of the index and even the middle fingers. There is frequently lessened sensation in the bone of the terminal phalanx of the thumb, and sometimes, too, of the first three metacarpals.

The stereognostic sense is always defective in the outer half of the hand. The patient cannot recognise the shape, consistence, or temperature of a thing which is placed in contact only with the three first fingers. The astereognosis is due to disturbances in superficial and deep sensibility and to motor changes in the first three fingers. In short, median paralysis, which is more serious from the point of view of function than paralysis of other nerves of the arm, makes the thumb and index finger—the most important fingers of the hand—absolutely useless, and also, though to a more variable degree, considerably affects the middle finger.

Sensibility of the ring finger is less affected, and frequently there is only diminished tactile and thermal sensation over its outer half.

In considering sensibility, it should not be overlooked that there may also be a vascular lesion, and in that case the intensity and distribution of the sensory as well as the vasomotor and trophic charges would, as we shall presently see, be greatly affected.

Vasomotor, Secretory, and Trophic Changes.—In many lesions of the median nerve the hand is discoloured, particularly the first three fingers. The skin is red or purple, dry and chapped, and the index finger is sometimes even cyanosed and cold, the nails being whitish or purplish.

There is no doubt that in most cases vasomotor, secretory, and caloric conditions should be attributed to a concomitant vascular lesion, which is of common occurrence owing to the proximity of nerves and arteries.

We shall refer to the importance of this complication later on. Examination of the pulse and blood pressure with Pachon's sphygmomanometer will deter-

mine whether there is an accompanying arterial lesion. Many cases are seen, however, without this complication in which the index finger is red and purple and the skin dry and chapped in the area of the damaged median nerve.

These symptoms are probably more marked in incomplete lesions of this nerve, as we shall show later. We have never come across very marked trophic changes in the skin and nails in severe damage to the nerve when unassociated with a vascular lesion.

Reflexes.—The *stylo-radial reflex* is often brisk, with marked contraction of the biceps and supinator longus; supination is exaggerated a little in consequence of the weakness of the pronators and flexors. The *triceps reflex* is also brisk. The *ulnar pronator reflex* is rarely obtained. The *flexor carpi radialis reflex* is absent.

Idiomuscular contraction.—Though at the onset this contraction may be strong, and even exaggerated, it soon grows weaker, and disappears entirely as atrophy increases.

On the other hand, percussion of the muscles in the thenar eminence at the level of the abductor brevis and opponens soon shows slowness of the contraction, with delay in release of the muscle again.[1] These muscles react to mechanical percussion as they do to the galvanic current.

When the atrophy is pronounced, percussion produces no contraction.

Recovery of Power of Movement.—We are inclined to think that in lesions of the median nerve the return of voluntary movement takes place in both complete and partial paralysis in very much the same way. But when the nerve has been completely divided the recovery of movement is so difficult and slow that we cannot say definitely how omplete recovery occurs.

Generally speaking, there is a slight indication of

[1] André Thomas, *Soc. de Biol.*, Jan. 22, 1916.

partial recovery of sensation long before power of movement returns, at least as far as the thumb and middle finger are concerned.

The stereognostic sense is very slow in being recovered.

The pronator and palmar muscles regain their functional activity first, then the flexors of the middle finger, and afterwards the flexor of the thumb.

Flexion of the index finger and opposition of the thumb are impaired for a very long time ; even after the lapse of close on two years we have no experience of cases of complete recovery.

The same precautions as were recommended to judge how much voluntary movement has been regained, and how real the recovery is in connection with the musculo-spiral nerve, should be taken in the case of the muscles supplied by the median nerve.

To test pronation the patient's elbow should be kept quite still whilst the observer carefully palpates the muscular part of the pronator teres just beneath the skin internal to the middle part of the supinator longus.

To test flexion of the hand careful palpation should be made both of the fleshy part of the muscles and of the palmar tendons at the wrist. The patient should be requested to carry out movements of flexion to see if the palmar muscles really contract.

In testing for the recovery of the power of movement the tendency of adjacent muscles to give prompt assistance should not be ignored.

It is a curious fact that the extensors of the hand and fingers have an action which at first might seem paradoxical in causing a movement of flexion of these segments.

This is particularly striking in associated lesions of the median and ulnar nerves.

Therefore, when muscles of the palm are examined the hand should be placed in prolongation with the forearm, after carefully correcting its inclination to extend.

Flexion of the fingers should be tried segment by segment after straightening the proximal phalanx, which is prone to over-extension during attempts at flexion of the fingers.

This hyperextension is also very striking in the thumb. When told to flex the second phalanx the patient first over-extends and abducts the thumb, and afterwards, sharply relaxing these muscles, brings the thumb back on to itself, and its second phalanx makes a slight passive movement of flexion on the proximal phalanx.

When the power of movement in the flexor longus pollicis has been regained to some extent, it is well to be on one's guard against the following manœuvre in judging of the amount of recovery of functional activity in the opponens and abductor brevis pollicis. The patient gets his thumb to the base of the ring or even the little finger in a semblance of opposition by scraping it, as it were, along the palm of the hand by means of the adductor and inner head of the flexor brevis pollicis, the second phalanx being somewhat flexed by the flexor longus.

B. *Partial Paralysis of the Median Nerve*

Our statistics show that partial or dissociated lesions of the median nerve are by far the commonest. Considering the gravity of the disturbances to function which total lesion of this nerve causes, this is a fortunate occurrence.

We have already dealt fully with the physiology of the muscles when the median nerve is completely divided, so that it is superfluous to speak in any detail about the method of examining voluntary movement in cases of partial lesion.

Motor Disturbances.—Methodical examination of the movements themselves should not be begun until accessory causes which might account for the loss of power have, as usual, been eliminated, such as articular lesions and cicatricial adhesions.

Flexion of the index finger is the most defective movement, though opposition of the thumb is very poor too.

There is very frequently imperfect flexion of the middle finger, and absence of this movement altogether in the second phalanx of the thumb.

The pronators and flexors of the wrist, however, are either not affected at all or very slightly paralysed.[1]

In cases of partial paralysis it is very easy to see how patients assist in placing the thumb in opposition, if the flexor longus is intact, by slowly moving it along the base of the other fingers.

Sometimes the opponens pollicis is only slightly affected, the abductor brevis being more so ; opposition, though only awkward and partial, is then, however, still possible.

Why are the flexors of the wrist and the pronators so relatively unimpaired when the flexors of the fingers are so frequently involved ?

The answer probably is that the branches intended for the flexor sublimis digitorum (particularly those fibres serving the index finger) and the branch supplying the thenar eminence are larger on account of the importance of the prehensile movements of the two first fingers.

But it must chiefly depend on the arrangement of these branches in the nerve-trunk and on the position of the wounds. The fibres of the flexors of the fingers are situated at the inner edge of the nerve : [2] the fibres for the thenar muscles occupy the anterior part of the nerve in its passage into the arm. In the forearm the fibres incline towards the radial side of the median nerve.

We have come across a case in which the median nerve was injured by the irritation of a callus surrounding a fractured humerus on the outer and posterior border of the nerve. The flexor longus

[1] Pierre Marie and Mme. Athanassio-Benisty, "Individualité clinique des nerfs," *Société de Neurologie*, March 15, 1915.

[2] Pierre Marie, Henry Meige, and Gosset, *loc. cit.*

pollicis and the pronators were involved, the other muscles being intact. Flexion of the index finger alone was imperfect, possibly on account of a lesion of the flexor profundus of that digit.

It is rare for the external border of the median nerve to be affected ; the internal border is, however, very often injured, and this accounts for the frequent paralysis of the flexors of the fingers in the many cases of penetrating wounds of the inner side of the arm.

Sensory Disturbances.—Sensory disturbances in partial lesions of the median nerve are slight. Hypæsthesia is the general rule ; anæsthesia is exceptional, and when present only affects the last two phalanges of the index finger. Tactile sensation and sensibility to heat and cold are most affected.

Bone sensibility, sense of position, and perception of pricks are merely diminished.

The stereognostic sense is defective in the outer half of the hand mainly because of the loss of functional activity in the muscles ; but as soon as the thumb can be drawn towards the palm of the hand, the patient can feel the temperature and consistence of any object put in his hand : he recognises its substance, and often succeeds in identifying it after some delay.

Vasomotor, Secretory, and Thermal Disturbances.—In partial paralysis uncomplicated by any vascular lesion, vasomotor and secretory troubles are considerable. The skin is cyanosed ; it is colder, and perspires more than the healthy part of the hand.

Electrical Reactions are very variable. Stimulation of the nerve-trunk itself often causes pronation and flexion of the hand.

The palmars and pronators which frequently react to the faradic current give a slow contraction with the galvanic current, and the same may be said of the flexor longus and of the thenar muscles.

Very careful electrical examination of these muscles

should be made: the thenar muscles often react well to a fairly strong faradic current when stimulation of the nerve-trunk itself, just above the wrist, only produces a single contraction of the interossei and of the adductor on account of the current being diffused into the ulnar, without causing any contraction of the thenar muscles themselves.

Reflexes.—The bone and tendon reflexes do not require any special description, though the ulnar pronator reflex and the flexor carpi radialis reflex are present, and may even be brisk. Idiomuscular contraction is usually strong, but it may be slower in the thenar muscles.

Recovery.—Recovery of sensory functions is quicker than recovery of motor power in partial lesions of the median nerve. Sensation to heat and cold is the last to return. The index finger is the slowest in recovering full sensation, especially in the terminal phalanx: for a long time the patient feels as if he had cotton gloves on when he touches an object. We have already referred to the gradual recovery of power of movement.

Signs of Recovery.—The median nerve may be considered quite restored when the patient can go through Pitres' test—*i.e.* when with his hand laid flat on the table, palm downward, he can scratch the wood of the table with his index finger without moving his wrist.

Clenched-fist sign (Claude).—A second sign of recovery of the median nerve is when the patient can firmly clench his fist with the fingers well flexed into the palm of the hand, and with the thumb firmly pressed on to the dorsal aspect of the second phalanx of the middle finger.

DIAGNOSIS

There is no disorder which can simulate total or partial paralysis of the median nerve barring actual lesions of the epitrochlear and thenar muscles, divisions of the nerve, cicatricial or tendinous adhesions

due to a wound, or fracture of the forearm or palm of the hand.

Apart from electrical examination, Pitres' tests for musculo-spiral paralysis are useful in diagnosing lesions of the median nerve.

The test of the hands crossed in an attitude of prayer would show whether the flexors were affected, as the index, middle finger, and thumb on the side where the median nerve is injured would not be able to clasp the hand perfectly nor to move freely.

The clapper test could be performed correctly, for only the extensors are involved in the movement.

Laying the back of the hand flat on the table and also laying it palm downward can be done perfectly.

But the same cannot be said of the clenched-fist test, as the middle and particularly the index fingers cannot be completely flexed into the palm, and this prevents the fist closing and the subsequent locking of the thumb on to the dorsal aspect of the articulations of the phalanges of the second and third fingers.

Briefly, the symptoms of paralysis of the median nerve are:—

1. *Inability to flex the phalanges of the index finger and the second phalanx of the thumb.*
2. *Difficulty in flexing the phalanges of the middle finger.*
3. *Defective opposition of the thumb.*

II. PAINFUL LESIONS OF THE MEDIAN NERVE

A very large number of lesions of the median nerve present an entirely different character, the predominant feature in such cases being *pain*. This painful type[1] of lesion was noticed during the American War of Secession by Weir Mitchell, who described its intensity and character under the name of Causalgia,[2] and attributed it to an ascending

[1] Pierre Marie and Mme. Ath.-Benisty, "Une forme douloureuse des lésions du nerf médian par plaie de guerre," *Académie de Médecine*, March 16, 1915.

[2] From the Greek καίω, "I burn," from which the word "caustic" is derived.

neuritis. According to Weir Mitchell, this form of neuritis might develop in any nerve and gradually attack all the other nerves of the affected limb : in addition, it might induce a cutaneous dystrophy, exposing, as it were, the terminal nerve organs subserving the sense of touch, and this would account for the burning pain complained of.

But in reality it has been proved by surgical intervention that this ascending neuritis develops most frequently in the region of the median nerve, and is due to lesions affecting that nerve exclusively.

In the lower extremity the same painful neuritis may be caused by the internal popliteal nerve. When a lesion of the ulnar nerve is accompanied by this intense pain, it is either due to an associated lesion of the median or else to injury of the brachial plexus affecting both ulnar and median nerves.

FIG. 25.—Painful type of wounds of the median nerve. Dorsal aspect of hand.

Pain in lesions of the musculospiral nerve is very rare.[1]

The **appearance of the hand** is important enough to be described in detail.

The patient holds his elbow in an attitude of flexion, with the wrist curved slightly inward, the hand always raised, the fingers extended, with occasionally hyperextension of the terminal phalanges (" cup hand ").

The thumb is in as perfect adduction and opposition as the condition of the thenar muscles will allow.

The hand is emaciated, atrophied all over, and refined and slender in appearance in contrast to the sound one.

The fingers are thin and tapering, the nails being

[1] Henry Meige and Mme. Ath.-Benisty, " Les formes douloureuses des blessures des nerfs," *Société de Neurol.*, July 1, 1915.

convex and pink. The skin, which is very fine, is white on the back of the hand and tends to be pink in the palm, with sometimes a deposit of deeper red pigment at the edges of the thenar eminence and the base of the fingers. Over the back of the fingers the skin is drawn, and it is creased and wrinkled transversely in the palm of the hand.

In many cases this *colour and appearance of the skin are confined to the region of the median nerve*, and are in sharp contrast with the neighbouring ulnar region.

Motor Disturbances.—These are usually slight. Total paralysis of all the muscles situated below the wound is of rare occurrence.

On the other hand, however, we have never seen this painful lesion without some weakness in movement. This is localised in the flexors of the fingers, particularly of the index finger and the thenar muscles, thus rendering the movement of opposition of the thumb rather difficult. The flexor longus pollicis, the palmar muscles, and the pronators may be involved in more serious cases.

The majority of cases are of moderate severity, and the impairment of movement then depends as much on the pain as on the weakness of the muscles ; so that between the attacks of pain, movement becomes so much easier as to allow the patient to grasp an object.

Secretory Changes.—Perspiration is diminished all over the outer part of the hand and in the first three fingers, though it is often increased over the dorsal aspect of the fingers. Sometimes there is no perspiration at all, making the skin so harsh and dry that it chaps and scales off.

In other cases perspiration is increased, and large beads of sweat appear all over the cutaneous area of the median nerve, which is in strong contrast with that of the ulnar region, where the skin is browner and drier.

Muscular Atrophy is less marked than in total paralysis, and is only apparent in the thenar eminence ;

but, to compensate for this, there is always some atrophy of the muscles of the limb situated above the lesion, and not supplied by the median nerve.

Sensory Disturbances.—A. *Subjective sensibility.*—By far the most striking symptom is pain. Very few patients state that the pain began at the time of the injury. Those who felt great pain when struck by the missile, and have suffered from it ever since, maintain that paroxysms of pain only came on a few days after the wound was inflicted.

Most patients had little pain in the early stages, and only complain of it from one to three weeks after the wound. The pain begins with formication and pricking at the ends of the fingers, followed by a burning and smarting sensation in the tips of the fingers and palm of the hand; for it is the hand only that is affected—pain is exceptional anywhere else.

This pain becomes more and more intense, and gives the patient no rest; the whole of the hand seems to smart horribly. When the patient describes the position of his pain more precisely, he refers it to a semicircle extending from the root of the thumb to the root of the index and middle fingers. The base of the index finger is particularly painful; then the patient complains of the tips of the fingers, especially the index and middle fingers. The thumb is occasionally very painful, but it may be as little affected as the little finger, which is always unaffected in pure lesions of the median nerve. Many patients point to the third interosseous space, the inner side of the middle finger, and the outer side of the ring finger as the most painful spots; *i.e.* where the terminal cutaneous branches of the median nerve end.

Different expressions are used to describe this pain. Some patients compare it to a *burning sensation* (*Causalgia* of Weir Mitchell), to pin-pricks, to a red-hot iron, to piercing, grinding, and dragging of the flesh; nearly all describe it as a throbbing pain, which recurs at fairly long intervals.

The *attacks of pain are paroxysmal.* The incessant

pain, which dates from the early months of injury, and which never ceases, even at night, suddenly increases in intensity owing to causes which the unfortunate patient delights in describing in the minutest detail.

An attack is usually brought on or prolonged by bending the hand down; so patients always keep it up. Exposure to air, dry heat (particularly a hot atmosphere), noise, sudden and bright light, deep breathing, coughing, sneezing, and laughing have the same effect. Emotional causes soon come into play, such as the sight of a wide, open expanse, an empty space, looking out of a window or descending a staircase, meeting a relative, the doctor's visit—in fact, any emotion is said to increase the pain. Then the condition becomes aggravated; the patients can no longer bear to see their fellow-patients play or to hear them laugh, because they are afraid of an accident; a comrade walking across a polished floor on crutches, the sight of anyone leaning out of a window, a workman on a roof, the flight of an aeroplane, or anything that suggests the possibility of a fall or giddiness, intensifies this pain.

Patients so affected are dismal, pale, and waxen, with intermittent fits of flushing, covered with perspiration, and martyrs to insomnia. They escape from noise, avoid society, and remain curled up in their beds or on a sofa in peculiar attitudes of defence, with the arm bent up and motionless, and the body often curved round towards the affected hand.

Shortly after the wound, patients notice that the burning heat of the hand is allayed, if only temporarily, by cold water, so they place their hand under the cold-water tap, with the result that soon they cannot get on without a wet bandage round their burning hand, which is hyperæsthetic. They will not allow the doctor to touch their hand until he has moistened his fingers, and they prefer to remove their own wet bandages or damp gloves by themselves. This phobia of dryness tends to increase. They cannot touch anything with their other hand without first wetting their

fingers. Even the epidermis of their feet becomes hyperæsthetic.[1] One of these patients asserted that he could not walk barefoot unless he wetted the soles of his feet at every fifth step ; and that his hair could only be combed after having been well damped. Occasionally attacks of pain are brought on by merely seeing someone touch a dry thing, such as a brick or a bar of iron. Rustling, sharp, and shrill noises, such as a knife-grinder makes, accentuate the pain.

Weir Mitchell reports a case of a patient with causalgia who could only walk when his boots had been filled with water, on the pretext that it deadened the shock of each step.

Walking, in fact, is a cause of pain to these patients, and often occasions more intense pain in the arm, elbow, and forearm than in the hand.

This pain reaches its height four or five months after injury ; then it gradually, and very slowly, diminishes in severity. Sometimes this phobia lasts as long as nearly two years after injury, and, though it may become less acute, patients still persist in wrapping their hands up in wet bandages.

Some of them state that they do not perspire as much as they used to do before their injury, and that this accounts for the distressing dryness. Others, on the contrary, say that the surrounding heat, which makes them perspire and increases moisture of their bodies, causes much discomfort, which reacts by pain in the affected hand.

Other patients, again, manage to do without water, being content with a dressing of cotton-wool ; but these are the less serious cases, in which perspiration is only slightly diminished or even exaggerated.

B. *Objective sensibility.*—The areas affected by these disturbances vary considerably. Sometimes the entire region of the median nerve is involved ; sometimes it

[1] A. Souques calls this phenomenon synæsthesalgia. See his paper entitled " Synæsthesalgia in certain forms of Painful Neuritis : its Treatment by Rubber Gloves," *Soc. de Neurol.*, May 6, 1915. *Rev. Neurol.*, July 1915, p. 562.

is only the index or middle finger, or the outer part of the palm of the hand; or, finally, the greatest loss of sensation may be found on the inner side of the middle and the outer side of the ring finger. .

Tactile anæsthesia, or hypæsthesia, generally speaking, is confined to the most painful spots. It should be tested with a wisp of cotton-wool, as the slightest pressure may cause hyperæsthesia. Pricking, in fact, occasions very marked hyperæsthesia in the area of the median nerve on the back and in the palm of the hand; whereas the pain may be diminished by deep pressure on the whole of the hand and fingers.

Hypæsthesia to heat and cold is always present, though heat often causes painful hyperæsthesia, which may be wrongly interpreted.

The sense of position and the sensibility of bone are more or less affected.

The stereognostic sense is greatly impaired, and sometimes entirely absent, even when there is enough movement left in the fingers to facilitate palpation. In other instances things may be recognised after a very long delay.

Pressure on the nerve-trunk is painful below, and very often above, the site of the wound.

The most sensitive spots are at the *junction of the upper extremity and the inner border of the thenar eminence* (point of election); at the *root of the second and third fingers*, and in their interspaces; along the whole course of the nerve in the wrist and forearm (in the middle of the radio-carpal joint, and the middle of the anterior aspect of the forearm); immediately internal to the tendon of the biceps; and, finally, along the internal bicipital groove, in the arm.[1]

Involuntary Movements.—Two other peculiarities are characteristic of this type of hand—slight irregular

[1] This hyperalgesia of the nerve can frequently be detected by the faradic current: when the electrodes are applied to the course of the median nerve in the forearm, intense pain is felt, in sharp contrast with the tingling sensations, which can easily be borne, occasioned by the same current on the adjacent skin.

tremor of the thumb and index finger in particular, but noticeable in all the fingers (Henry Meige). It increases when the patient tries to move his fingers, and when the attacks of pain come on.

It gradually diminishes as the fingers ankylose, or as improvement takes place. But in severe cases permanent involuntary movements, which are sometimes constant and sometimes variable, occur, and may persist for months.

Such a case will present involuntary and sometimes clonic movement, which lasts for a long time, and consists in flexion of the third and fourth fingers on the metacarpals. This movement will then be followed by a jerking lateral motion of the middle towards the index finger.

In other cases the thumb presents movements of laterality or opposition ; or the index finger moves towards the palm of the hand.

Thermal Symptoms.—The patient complains that his hand is always hot, and at times burning ; its temperature is, in fact, a few degrees higher than that of the other hand, at some moments at least. Less commonly, he complains of attacks of sudden chilling of the hand. The blood pressure, measured by Pachon's sphygmomanometer at the time of a paroxysm, is increased by half a degree or a degree in comparison with the sound side, but, generally speaking, even in young people, the tension is pretty high (18–19–22).

Changes in the Reflexes.—The tendon and osteo-periosteal reflexes of the upper extremity are usually brisker than on the sound side, but, on account of the pain they cause, their action is not easily examined. We have, however, come across cases in which they were diminished, or even completely abolished.

Idiomuscular contraction is also brisk.

EVOLUTION

Improvement in the pain is, as we have already said, spontaneous.

In *voluntary movement* there is either no change at all or only very slow progress. Frequently this function gets even worse as time goes on.

Attitudes of defence, *e.g.* holding back the whole of the arm in flexion—a favourite position with patients—cause irreducible ankylosis, with very pronounced retraction and crowding of the fingers, which render the limb, and particularly the hand, quite useless (see figs. 26, 27, 28).

Atrophy of the hand is progressive and generalised.

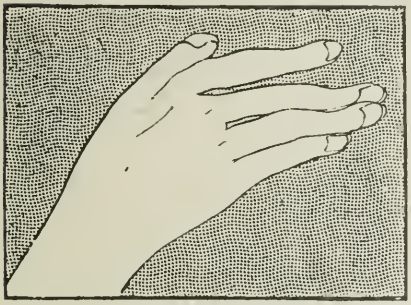


FIG. 26.—Painful type of wounds of the *median nerve*; condition of hand twenty months after injury. Marked ankylosis and deformity of the terminal phalanges and nails.

FIG. 27.—Aspect of the hand in the painful type of wounds of the *median nerve*, showing deformity of fingers and nails. Ankylosis of wrist in hyperflexion.

The skin is finer; the epidermic elevations having disappeared, the deeper layers appear shrunk; the skin becomes more and more creased and wrinkled on the palm of the hand, but stretched over the dorsal aspect of the fingers. It is red and blotchy in places, especially at the base of the first three fingers. Sometimes, though this is rare, there is a hyperkeratosis of the palm at the base of the fingers, which become increasingly thinner and more tapering.

The nails are bulging, thickened, and streaked with transverse or longitudinal striæ. Sometimes they grow

out of all proportion and curve inward ; the skin of the nail bed grows up and attaches itself to the nail, which, as it grows, drags it along with it and causes still more pain (fig. 29). The tips of the fingers become swollen and show slight clubbing, the sides of the nails tending to lap over the grooves in which they are embedded.

The hand becomes so fine and drawn in appearance that it looks as if atrophy had spread to the bones. Skiagrams show very marked decalcification of the phalanges (Sicard's " osteoporotic hand ").

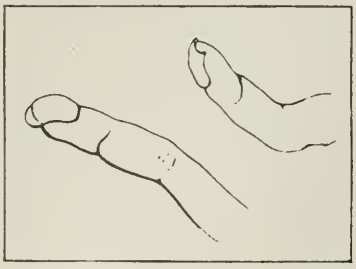


Fig. 28.—Appearance of fingers and nails in the painful type of lesions of the *median nerve.*

Fig. 29.—Catenulate and striated nails in the painful type of lesions of the *median nerve.*

PATHOGENY

Although it is intended in the second section of this work to deal with anatomical lesions of the nerves, their restoration and treatment, we shall now describe the results of surgical intervention from the anatomical and therapeutic point of view, and the different varieties of treatment that have been used in cases of causalgia of the median nerve ; for this information will help to elucidate the pathogeny of this painful lesion, which is so unlike other forms of paralysis.

Gosset's operations for causalgia in the hand, performed on several patients in Prof. Pierre Marie's wards, have shown that the median nerve is only slightly affected.

At one time it might be slightly swollen or indurated and greyer for some distance ; at another it was found to be constricted by a more or less tight fibrous band, or the nerve might be œdematous, red, and congested.

In such cases as these the surgeon merely freed the nerve, and the course of the disease was not affected by the operation. In another case, in which the median nerve did not respond to stimulation by an electric current during the operation, Prof. Pierre Marie had the swelling on the nerve torn apart; the pain was better on the following day, and in a week's time had almost entirely disappeared.

Histological examination is indispensable for the study of the changes that occur in the nerve, but, as resection is not required in this type of lesion, this examination was not undertaken, and the histological changes in the nerve are still unknown.

Various forms of treatment were tried. First, the nerve was freed, but without much result ; then, palliative treatment, such as hot air, hot baths to the arm, the constant current, and ethyl-chloride spray, but all with no effect.

When causalgia could be traced to a lesion of a nerve-trunk, treatment was applied to the nerve-trunk itself.

Some neurologists have made use of *radio-therapy*. Cestan and Paul Descomps did so systematically in the Neurological Department of the XVII. Region [1] with marked success in painful lesions of the median nerve ; improvement was less pronounced in the case of the sciatic nerve, perhaps because the latter is less easy to get at. The technique adopted was as follows :— 1 milliampère with a hard tube (the hardness of the tube is important), Rays VIII.–IX. Aluminium filter of 1 mm. The anode, at a distance of 20 cm. from the skin, applied for a quarter of an hour once a week.

In Prof. Pierre Marie's ward, Dr Grunspan has also

[1] *Presse médicale*, November 25, 1915.

been successful in the use of radio-therapy for these painful nerve-lesions.

But as this treatment could not always be relied upon, other methods had to be resorted to.

In very severe cases, Prof. Pitres, reviving an old practice of Weir Mitchell's, performed *section of the nerve itself* in the case of the sciatic, as the median nerve was too important from a motor point of view to have section performed. In a painful lesion of the internal popliteal, section of the posterior tibial was made at the level of the internal malleolus, *i.e.* at a point where this nerve had already supplied its main muscular branches. The operation relieved the pain in the sole of the foot.

Sicard, however, tried to perform section of the median nerve at 5 cm. above the lesion, suturing it at once to prevent injury to its function, but with only partial relief of the pain.

He next injected *alcohol into the nerve-trunk*, with good results. The injection should be made 2 or 3 cms. above the seat of lesion and into all the branches of the painful nerve-trunks, in case of a painful lesion in a nerve plexus. A general anæsthetic should be given, and, after exposing the nerve and freeing it from the surrounding cicatricial tissue, the injection should be made with a fine syringe of a 1-c.c. cube of sterilised alcohol at 60 per cent. It should be sterilised by tyndallisation in closed ampoules to preserve the strength of the alcohol.

When the injection has been properly carried out an œdematous swelling forms and the nerve-trunk assumes a characteristic pearly-white colour at once.

Alcohol at 60 per cent. only interferes for a limited period with the function of motion, which returns in from six to eight months. The relief of pain resulting from this treatment is quite remarkable.[1]

[1] Sicard, "Traitement de la névrite douloureuse du médian par l'alcoolisation tronculaire sus-lésionnelle," *Soc. Méd. des Hôp.*, July 9, 1915; "Traitement de certaines algies et acrocontractures rebelles par l'alcoolisation nerveuse locale," *ibid.*, December 17, 1915.

Finally, Leriche adopted an ingenious method. He tore asunder the periarterial sheath of the sympathetic in a case of lesion of the brachial plexus with excruciating causalgia and vasomotor changes which pointed to a lesion of the axillary artery, the result of the operation being that the pain was greatly relieved.

Causalgia was first observed in 1813 by Alex. Denmark, then by Paget, and finally by Weir Mitchell, who has given a remarkable description of it. He attributed the pain to an inflammatory process beginning at the seat of injury and advancing up the nerve to the spinal cord ; it involved the adjacent nerves, caused a kind of atrophy of the skin of the hand, making it " glossy " and exposing and setting on edge its sensory areas—hence the cutaneous hyperæsthesia complained of. It is quite certain that in the term " causalgia " Weir Mitchell included both the more or less acute pain met with in vascular lesions associated with injuries of the peripheral ends of the nerves, and also the causalgia due to lesion of the trunks of the median and internal popliteal nerves, in the course of which the " glossy " skin characteristic of vascular lesions is rarely seen.

The pathogeny of these painful lesions, therefore, still needs investigation.

At a meeting of the Paris Neurological Society on 1st July 1915, when Henry Meige and ourselves reopened a discussion on painful nerve lesions and their treatment, we drew attention to two facts :—

1. The *structural peculiarity of the area affected by causalgia* (palm of the hand, sole of the foot).

2. *Congested appearance* of the nerve-trunk noticed by several surgeons and neurologists during operations for these painful lesions of the median and sciatic nerves. On one or two occasions Gosset saw some fine arterioles penetrating, and as it were separating, the nerve.

We have also pointed out that the median and sciatic nerve-trunks possess not only a special peri-

pheral area but also a most important vascular system of their own.

We were afterwards led to study the part played by the sympathetic system by reason of the throbbing nature of the pain, its intensity and analogy with the different visceral neuralgias, its influence on the mind, and careful study of the histological conditions of the skin and its adnexa (*i.e.* sensory terminals, sweat and sebaceous glands, capillaries of the papillæ of the skin, etc.).

It is easy to define the share this system takes in the case of the sciatic, as it is known that most of the vasomotor fibres of the lower extremity accompany this nerve.

This fact has been confirmed by Claude Bernard's classical experiment, in which he performed section of the sciatic nerve on a rabbit and afterwards noticed active vasodilatation of the blood-vessels of the foot with local rise of temperature.

Physiological experiments on the upper extremity have not been as numerous or as definite.

It is known that a sympathetic plexus surrounds the axillary artery and its collateral and terminal branches, but less is known about the sympathetic fibres which follow the nerve-trunks of the limb. Nor is it known which nerve of the arm has most of these fibres.

At the meeting of the Neurological Society on 6th January 1916, Leriche, in recommending the treatment we mentioned above, raised the question whether his case was not probably one of neuritis of the periarterial sympathetic, as the vascular lesion was so apparent and pronounced.

This ingenious supposition should be correct as regards the acute symptoms which are sometimes present in vascular lesions associated with lesions of peripheral nerves.

But how can we explain either the painful forms of greater severity following lesions of the median nerve in the absence of any vascular lesion, and in which the

blood-pressure is by no means diminished, or cases of causalgia due to injury of the median nerve in the forearm and wrist at a spot where no important artery accompanies the nerve?

Neither does the hypothesis of a sympathetic periarterial neuritis explain the painful lesions of the great sciatic nerve caused by a wound of the thigh or buttock. Our present view is that in the obstinate types of purely nervous origin—the only types under consideration—we are concerned *with inflammation of the fibres of the sympathetic system following the nerve-trunks themselves* and supplying the glands, capillaries, and nerve-endings of the different layers of the skin.[1]

Do these sympathetic fibres which, when irritated, give rise to all the symptoms described above, prefer the nutrient artery of the median nerve; do they mix with the other fibres of the nerve; or do they prefer the course of the sensory fibres, as seems to be proved in the case of the sciatic nerve? We cannot say definitely.

Then, having taken for granted irritation of the fibres of the sympathetic, how can we explain the causalgia and the vasomotor and trophic changes which accompany it; and what can the nature of the irritation be which brings about such important changes in the structures of the hand situated at such a distance from it?

A long time ago histology showed us that the sensory nerve-fibres of the skin have special end organs called corpuscles or organs of touch, named after those authors who first described them, viz. Pacinian corpuscles, Ruffini's endings, Meissner's tactile corpuscles, organs of Golgi-Manzoni, Dogiel, etc.

The precise function of each of these corpuscles or organs is not known, nor whether they bear special relation to touch, pain, heat, etc.

It has been accepted, somewhat arbitrarily, that

[1] Henry Meige and Mme. Ath.-Benisty, "Les signes cliniques des lésions de l'appareil sympathique et de l'appareil vasculaire dans les blessures des membres," *Presse médicale*, April 6, 1916.

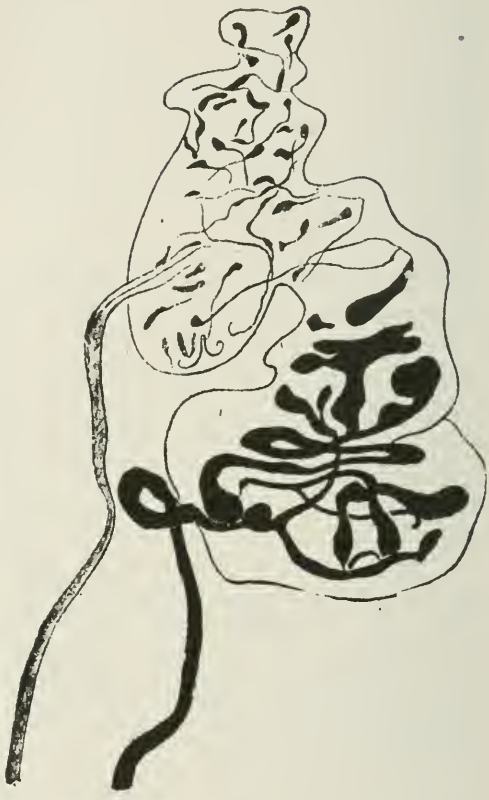
the painful sensations were taken up by the free

B A

FIG. 30.—Nervous structure of a Pacinian corpuscle. (After Timofeew.)

A, Central myelinated fibre of spinal origin.

B, Organ of Timofeew' formed by a network of amyelinated fibrils, probably of sympathetic origin.



B A

FIG. 31.—Nervous structure of a Meissner's corpuscle. (After Ruffini.)

A, Myelinated fibre ending in a nervous expansion.

B, Above. Organ of Timofeew connected with a non-myelinated fibre.

sensory terminations in the skin. But, as a matter of fact, the question still remains very obscure, and

further physiological investigation is required in support of this.

However this may be, it should be remembered that these corpuscles are particularly numerous in the palm of the hand and sole of the foot.

The corpuscles described by Meissner, as essentially tactile, only occur in the hand and foot, while the Pacinian corpuscles, which are much more widespread, are very numerous in these situations.

Rauber, who counted 1051 in half the body, apportioned 414 to the hand, 275 to the foot, and only 362 to the remainder of the superficial integument.

A few details of the histological structure of these corpuscles should be referred to.

In 1895, Timofeew first described a peculiar arrangement of the nervous tissue in some Pacinian corpuscles, in which around the large central nerve fibre, representing the termination of a sensory fibre, he found a net-work of nerve fibrillæ encircling the end organ without anastomosing with it (fig. 30).

FIG. 32.—Cap of nerve-fibrils covering a capillary loop. (After Ruffini.)

Other histologists have noticed this same arrangement in other sensory corpuscles, and Ruffini noted it around Meissner's corpuscles (fig. 31).

All sensory corpuscles of the skin, therefore, have two kinds of fibres: one central and the other peripheral, the latter encircling the former in an irregular manner.

Ruffini concludes from his own histological observations and those of his predecessors, that, in addition

to the terminal nerve-fibre contained in the sensory corpuscles of man, *there is a network of fibrillæ with special morphological characteristics connected with this termination by continuity or contact.*[1]

Moreover, Ruffini considers too that the central termination in which a large myelinated nerve-fibre ends might arise from the cerebro-spinal nerve-fibres, whilst the peripheral fibrillæ, springing from a fine fibre, not always provided with a visible myelin sheath, might originate in the sympathetic.

We are therefore inclined to think[2] that an irritation of the nerve-trunk in the wound is transmitted by the centrifugal fibres to the sensory corpuscles of the skin. These corpuscles being continually irritated in this manner, would cause the intense pain which occurs at times spontaneously, and at others is due to external causes, such as contact, jarring, variations in temperature or in the circulation of the limbs, or to emotion of any kind. These causes affect the vascular tension generally, and particularly the groups of small capillaries which abound in the papillæ of the skin, the glomeruli of the glands, and the sensory corpuscles.

Finally, anything that can modify vascular tension in the body reacts on the nerve lesion by impressions varying in intensity and frequency on the fibres of this nerve. These fibres are neither motor nor sensory, but take a centrifugal course, and are distributed to the various layers of the skin and subcutaneous tissues.

Some of these fibres have probably a trophic action. As in the case of vasomotor fibres, their irritation causes active vasodilatation in a succession of waves, considerable rise in local temperature, and atrophy and impairment of nutrition of all the tissues, such as might be brought about by an increase in local combustion.

The trophic state of the skin, subcutaneous cellular tissue, bones, and nails is affected in a very different manner from what is seen subsequent to arterial lesions due to constriction or occlusion.

[1] Testut, *Anat. descriptive*, tome iii. p. 314.

[2] Henry Meige and Mme. Ath.-Benisty, *Presse médicale*, April 6, 1916.

In short, the vasomotor and trophic changes following the vascular lesions, which we shall describe further on, are a kind of necrobiosis, and take the form of a diminution in the vitality of the tissues; whilst the trophic and vasomotor changes in the painful type correspond to an overactivity of the process of nutrition and a disturbance in the growth of the tissues.

Of what nature, then, is this nerve lesion? In most cases the onset of causalgia is not connected with suppuration of the wound, which has frequently healed in a few days and has not suppurated at all.

But supposing there is only a moderate amount of suppuration from the wound, the nerve itself may still have an inflammatory focus, for its tissue, as far as can be seen during operations performed on a number of wounded, is œdematous, red, and congested in appearance. Later on there may also be an induration of a few centimetres in extent, which reduces the diameter of the nerve, or else there may be thickening.

Does not this appearance suggest subacute or chronic inflammation, on which interstitial sclerosis has supervened?

This is merely a hypothesis, as we are still unable to explain the real morbid process in the lesion.

However this may be, the motor fibres are little affected, and paralysis is rarely serious; but the lesion irritates the nerves, as evidenced by the tremor, involuntary movements, and constant twitching of some fingers.

The fibres most affected are the vasomotor, secretory, and perhaps also the trophic, which provide nutrition for all the tissues supplied by the nerve.

In conclusion, we consider the intense pain and the vasomotor and trophic changes accompanying it due to what appear to be trivial lesions of the nerve-trunks, probably inflammatory in nature.

We think that these lesions particularly affect the vasomotor, secretory, and trophic fibres of all the tissues served by this nerve. The stronger fibres, such as the motor, are only slightly interfered with by

this irritative process, as may be seen by the paresis, tremor, and twitching ; other fibres, such as the secretory, may in some cases be entirely destroyed, but the great majority undergo a kind of irritation, which reacts on the capillaries of the papillæ of the skin, on the sensory corpuscles, the skin-glands, the subcutaneous cellular tissue, the joints and bones, etc., resulting in the complex of symptoms described by Weir Mitchell under the term " Causalgia."

The course of these centrifugal fibres referred to is either the periphery of the nerve-trunk or along its sensory fibres, and probably also to a great extent that of its most important nutrient arteries.

Bourguet's recent investigations (*Société de Biologie*, April 5, 1915) have shown that the median nerve receives a branch from the brachial artery, which penetrates it and accompanies it for a long distance down the arm. In the forearm the ulnar supplies it with an important branch, *the artery of the median*, which accompanies the nerve along its whole course. Finally, the branches of the median nerve in the hand are supplied by a number of arterioles, independent of those already mentioned.

However, considering the frequent anastomosis with fibres of the sympathetic, we are inclined to hope that, in some cases, Leriche's operation, which consists in tearing apart the periarterial sympathetic sheath, will be beneficial to the disorders of the sympathetic fibres contained in the nerve-trunk itself.

CHAPTER IV

THE ULNAR NERVE

JUDGING by the functions of the muscles to which it is distributed, the ulnar nerve would appear to be the inner branch of the median: injury to it causes disturbances to movement and objective and subjective symptoms; the latter, though not so marked as in the case of the median, are nevertheless characteristic and fairly constant.

ANATOMY

The ulnar nerve arises from the inner head of the median in the axilla behind the lower border of the pectoralis minor (see fig. 16). It lies in close relationship to the axillary artery and median nerve on its outer side, and to the axillary vein and internal cutaneous nerve on its inner side.

In the arm the ulnar nerve follows the internal bicipital groove between the biceps in front and the brachialis anticus behind. Here the nerve-trunks can be easily distinguished and palpated, especially in thin subjects; they can be rolled about under the aponeurosis covering the arm, the skin, and subcutaneous cellular tissue.

A little above the internal condyle of the humerus the nerve, passing backwards, pierces the inner head of the triceps and, after passing through this muscle, reappears on the posterior aspect of the elbow in the groove between the internal epicondyle and the olecranon, where it can again be felt on palpation.

It then passes on to the anterior aspect of the forearm between the two heads of the flexor carpi ulnaris.

At about 8 cm. below the bend of the elbow (fig. 17) it is joined by the ulnar artery. For about 10 cm. it is covered by the thick layer of the epitrochlear muscles, and lies on the flexor profundus digitorum: then it becomes more and more superficial.

It is only in the forearm that the ulnar nerve gives off important

branches, viz. *muscular branches* near the elbow, to the *flexor carpi ulnaris* as it passes between its two heads, and a little lower at the upper third of the forearm to the *inner half of the flexor profundus digitorum.*

The *dorsal cutaneous branch* arises four fingers' breadths above the wrist; it runs downwards, inwards, and backwards, and it supplies the skin on the inner side of the back of the hand, the dorsal aspect of the little finger, and part of the dorsal aspect of the ring and middle fingers (fig. 33).

At the wrist the ulnar nerve, which is now very superficial, is

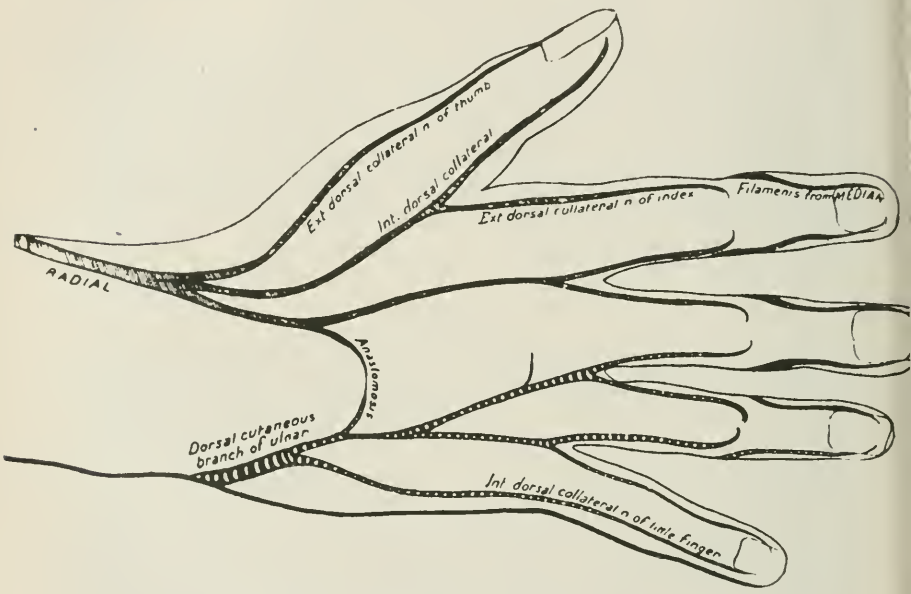

Ext dorsal collateral n of thumb
Int. dorsal collateral
Ext dorsal collateral n of index
Filaments from MEDIAN
RADIAL
Anastomoses
Dorsal cutaneous branch of ulnar
Int dorsal collateral n of little finger

FIG. 33.—Distribution of the nerves on the dorsal aspect of the hand.

on the outer side of the pisiform bone, and immediately below this bone divides into its two terminal branches.

The *superficial branch* supplies the skin of the hypothenar eminence, and furnishes the last three collateral nerves of the fingers (fig. 33). It gives a small muscular branch to the *palmaris brevis.*

The *deep branch*, which is muscular, passes between the abductor and flexor brevis minimi digiti, and runs very deep down in close relationship with the metacarpals and interossei, accompanied by the deep palmar arch. It sends motor filaments to all the muscles of the *hypothenar* eminence, to the *two inner lumbricales*, to *all*

the palmar and dorsal interossei, and then ends by supplying the *adductor pollicis* and the *inner head of the flexor brevis pollicis*.

Anastomosis of the ulnar with the median nerve occurs twice—in the hand by means of its superficial palmar branch, and inside the flexor brevis pollicis muscle by means of its deep palmar branch.

PHYSIOLOGY

The ulnar nerve supplies most of the muscles of the hand.

The *flexor carpi ulnaris* is one of the flexor muscles of the wrist; it inclines it to the inner side, but imparts no movement of adduction. The opinion of Duchenne (of Boulogne) is quite correct.

The *two inner heads of the flexor profundus digitorum* flex the

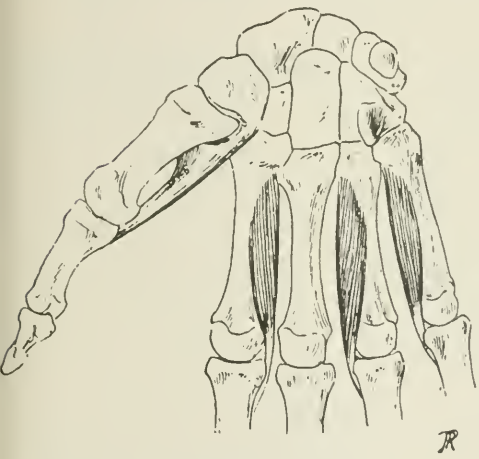


Fig. 34.—Palmar interossei.

Fig. 35.—Dorsal interossei.

second phalanges, and particularly the terminal phalanges of the ring and little fingers.

A brief description of the anatomy will make the physiology of the interossei clearer.

The interossei are seven in number: four dorsal and three palmar; figs. 34, 35, and 36 show their insertions.

The lower tendons of both these sets of muscles are attached to the extensor tendon.

The dorsal interossei, which are the most important, have a lower tendon which is divided into two portions. The short portion is attached to the upper extremity of the first phalanx on the side corresponding to the metacarpal bone, where the insertion of this muscle is more widespread; the long portion expands into a long, wide membrane which ends in the tendon of the corresponding extensor from the first phalanx to the terminal.

With this long head of the terminal tendon of the interossei the tendon of the lumbricales unites.

The interossei are very powerful muscles. Their two chief functions are: (1) they flex the first phalanx on the metacarpal and extend the second and terminal phalanges (fig. 37); (2) they abduct and adduct the fingers. The movement of adduction requires much energy, especially if the fingers are in extension.

The *lumbricales* also flex the first phalanges and extend the other two. The first lumbrical is the only one that exercises any lateral movement, and is only a very weak abductor of the first phalanx of the index finger.

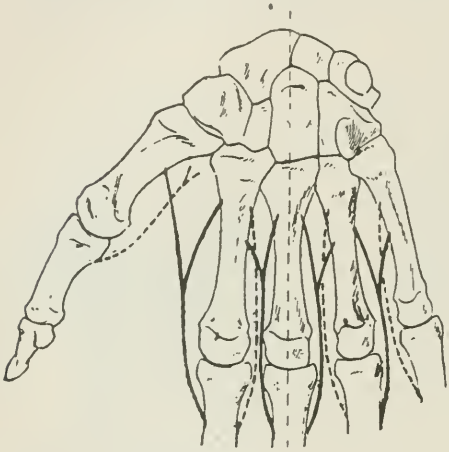


FIG. 36.—Plan of insertion of the interossei.

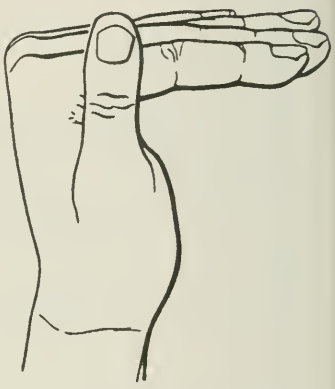


FIG. 37.—Principal movement of the interossei muscles (flexion of the first phalanx, extension of second and third).

There are four *muscles in the hypothenar eminence: palmaris brevis, abductor minimi digiti, flexor brevis minimi digiti, and opponens minimi digiti* (fig. 19), but they are insignificant in comparison with the corresponding muscles of the thumb.

The *palmaris brevis* is merely superficial, and, like most of the cutaneous muscles not affecting voluntary movements, does not call for further comment.

The *abductor minimi digiti* inclines the little finger to the ulnar border, drawing it away from the centre of the hand: some anatomists call it the adductor because it draws the little finger toward the median line. It assists the *flexor brevis minimi digiti* in flexing the proximal phalanx of the little finger on its corresponding metacarpal bone.

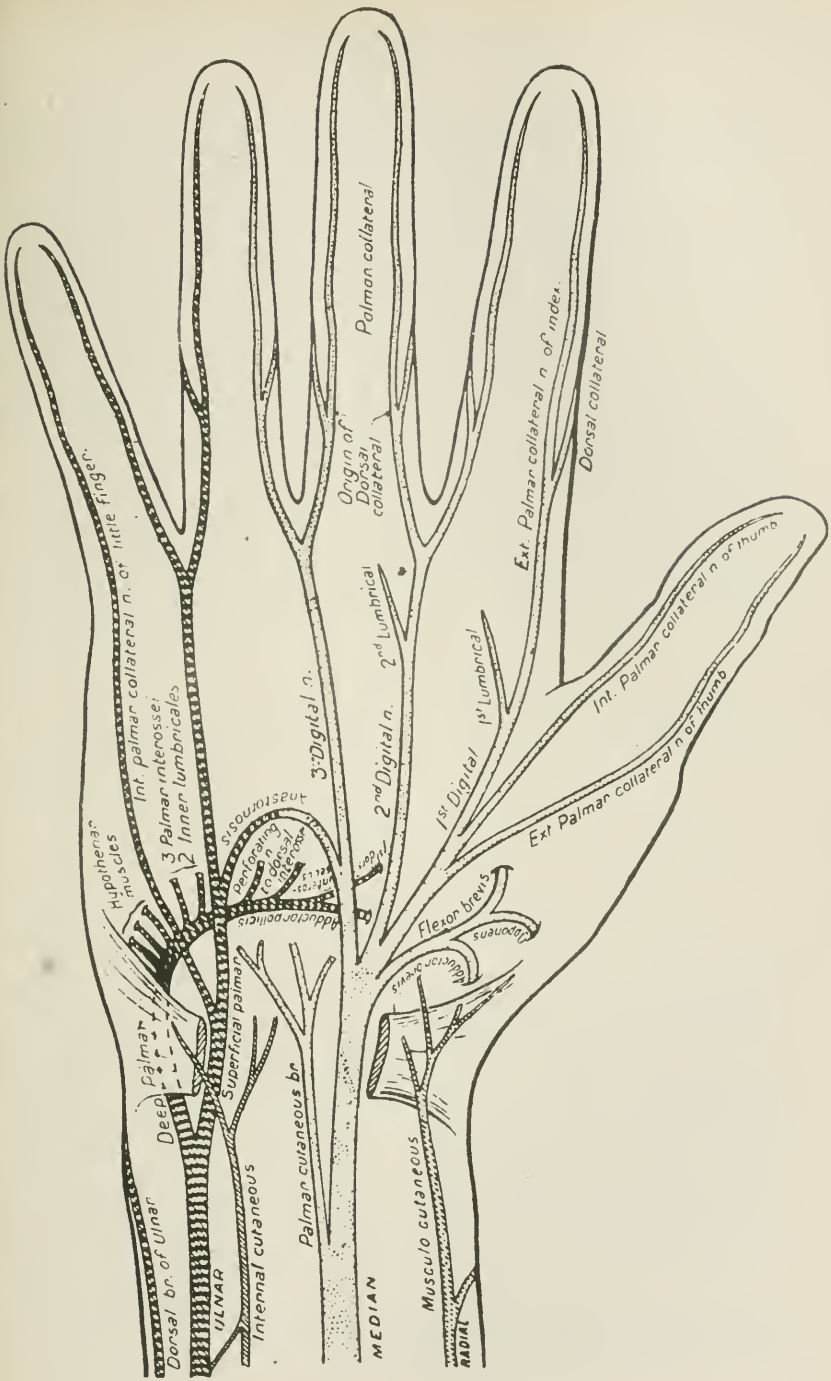


PLATE III.

Nerves of hand and fingers, palmar aspect.
(After Henry Meige.)

The *opponens minimi digiti* draws forwards and outwards the fifth metacarpal bone, and the little finger with it, without imparting any visible rotatory movement to that bone.

Finally, the ulnar nerve supplies the *adductor pollicis* and the *inner head of the flexor brevis pollicis*, sometimes called the *two inner sesamoid muscles of the thumb*, which are analogous in action. The adductor slightly flexes the first phalanx, tilts it on its inner border, and at the same time gives it a movement of rotation on its long axis—the opposite of the movement made by the outer sesamoids ; with the result that the first metacarpal is drawn outwards and a little in front of the second. The adductor, in addition, extends the second phalanx of the thumb on the first, so that when this muscle is fully contracted the first metacarpal is on a level with the second, the second phalanx of the thumb in extension, and the proximal closely attached to the outer border of the metacarpo-phalangeal joint of the index finger.

So we see that the second phalanx of the thumb is extended both by the extensor longus pollicis and by the external and internal sesamoid muscles of the thumb, which send expansions to the extensor tendon.

Clinical Types

We will subdivide these, as in the case of the median nerve, into—

I.—Total paralysis.
II.—Partial paralysis.

I.—Total Paralysis

The soft parts on the internal aspect of the arm are most commonly wounded a few centimetres above the internal condyle of the humerus.

Appearance of the Hand.—As the ulnar is the chief nerve which supplies the muscles of the hand, the appearance of the latter should be carefully examined. First, there is wasting, which is sometimes very marked, of the first dorsal interosseous space—an inseparable sign of a lesion of the ulnar, even more so in our opinion than *claw-hand*, though that is also very frequently present.

The claw-hand shows degrees of intensity which cannot always be explained by the situation of the

lesion and the extent of paralysis of the flexor profundus and interossei.

We should take into account the assistance rendered by the median nerve in supplying the inner fasciculi of the flexor profundus, and also a certain degree of retraction of tendons commonly seen in the deep flexors of the ring and little fingers, although they ought to be completely paralysed and limp.

When the ulnar nerve has been divided or severely injured in the middle third of the arm, though the clawing may be only slight, careful examination will show that it does exist. It consists of a more or less marked hyperextension of the first phalanx of the ring and little fingers, with a moderate degree of flexion of the second phalanx on the basal phalanx and a very slight flexion of the terminal on the second phalanx.

Sometimes the clawing which can be clearly seen in the little finger hardly exists at all in the ring finger ; at other times the middle finger is also more or less clawed.

Even in this incomplete form, the clawing cannot be overcome, and the little finger can never be completely extended.

The other characteristic features of the "ulnar hand" are the following :—

The little finger stands off from the ring finger in a very marked way ; the ring finger not so much so from the middle ; and the middle still less so from the index.

It is so important to know the exact *appearance of the hand* in ulnar paralysis that we desire most emphatically to call attention to the very great atrophy of the interossei, which sometimes gives the hand a *skeleton-like appearance.*

This wasting of the muscles is particularly marked in the first dorsal interosseous space. On the palmar aspect atrophy of the hypothenar eminence is considerable ; and firm palpation here gives one the impression of a hollow rather than an eminence. The amount of atrophy of the adductor of the thumb

should be gauged by look and touch at the inner part of the thenar eminence; the superficial parts are flabby, and sink down into large soft folds in the triangular space between thumb and index finger.

Motor Changes.—*Paralysis of the flexor carpi ulnaris.*—The patient should be asked to flex his wrist hard and to resist any attempt at extension; inspection and palpation will show that the tendons of the palmars only contract under the skin; the tendon of the flexor carpi ulnaris, which is strongly contracted on the sound side, is quite limp on the paralysed one.

Paralysis of the flexor profundus of the little and ring fingers.—When the patient is told to fold his fingers well into the palm of his hand to show his fist, it will be seen that this flexion, though normal in the index and often in the middle fingers, is quite imperfect in the ring and little fingers, which can only bend at the first inter-phalangeal joints. The flexion produced at the metacarpo-phalangeal and second inter-phalangeal joints is only the result of the influence exerted on all the segments of the finger when the flexor sublimis contracts—an influence more apparent in the little than in the ring finger.

The weak flexion of these two fingers is better demonstrated if the index and middle fingers are kept in extension.

At other times, this almost passive flexion of the proximal and terminal phalanges is very decided, and the fingers seem to close entirely—but in appearance only; the power of resistance is nil in the terminal phalanx, and is generally greatly diminished in the other two phalanges, though the flexor sublimis may be intact.

Paralysis of the hypothenar muscles makes it almost impossible to flex the proximal phalanx of the little finger separately on its metacarpal, whilst the other fingers are kept in extension.

Further, movements of abduction, and especially adduction, of the first phalanx of the little finger are

absolutely impossible. In spite of all the patient's efforts the hypothenar muscles make not the slightest attempt at contraction.

Paralysis of the interossei and of the inner two lumbricales causes the posture known as "claw-hand." The interossei being deprived of their nerve supply, there is no limit to the remarkable tonicity of the flexors, and the two terminal phalanges are strongly flexed. But the flexor profundus is also totally paralysed in a severe lesion of the ulnar nerve in the arm; flexion of the second phalanx on the first is only performed by the flexor sublimis, which, as it contracts, draws the terminal phalanx with it. At the same time the extensor of the fingers, having no antagonist for the proximal phalanx, hyperextends it.

Though all the interossei may be paralysed, there is only clawing of the fourth and fifth and sometimes of the middle fingers. This is explained by the immunity of the first two lumbricales, which are supplied by the median, and whose action is the same as that of the interossei, as far as flexion of the proximal phalanx and extension of the two others is concerned. Sometimes the lumbrical of the middle finger is supplied by the ulnar nerve, and this would explain why this finger is frequently clawed; but more often the middle finger is passively flexed by the movement of the fourth and fifth fingers.

The middle finger is in fact much less independent than the index. Moreover, extension of the second and terminal phalanges of the index finger, even though normal, is not nearly so strong. Patients with injury of the ulnar nerve complain of this weakness in their index finger. They cannot place a thing on a table with the tip of this finger; they have trouble in writing; they are less adept in doing certain things in connection with their trade which involve energetic extension of the last two phalanges.

When a patient is told to flex the first phalanx of the fingers and extend the other two, he can do it with his index and middle fingers by means of the

lumbricales, but his fourth and fifth fingers cannot carry out this movement. Their proximal phalanx becomes hyperextended, whilst the second and terminal keep flexed.

The lateral movements of abduction and adduction of the fingers are absolutely lost in the case of the little, ring, and often in the middle fingers; they are much diminished in the case of the index, these movements being due to contraction of the first and second lumbricales and the extensor of the fingers. Indeed, forced extension of the first phalanx is accompanied by abduction of the fingers, which is very marked in the index and little fingers, probably due to the action of their own extensors.

This power of abduction of the extensors is very remarkable in the little finger, which, as we have shown, is always in abduction[1] in total paralysis of the ulnar nerve.

Paralysis of the adductor pollicis and of the inner head of the flexor brevis pollicis results in disturbances to prehensile movements, which have been drawn attention to by Duchenne (of Boulogne), and recently investigated by J. Froment.[2]

If a patient is asked to grasp any object with his thumb and index finger, such as a folded newspaper, and told to hold it tight, he vigorously flexes the second phalanx of the thumb and presses its tip awkwardly against the outer margin of the first phalanx of the index finger. If anyone pulls at the newspaper, he cannot retain his hold on it, however much he tries; he cannot succeed in grasping the object with his thumb by extending the second phalanx, as is done normally. This is Froment's *sign of the newspaper*. In the same way, if the patient is told to press his extended thumb firmly against his index finger, the

[1] To realise the strength of abduction of the index and little fingers, extend the fingers forcibly on the metacarpals, taking care not to give them any additional movement.

[2] J. Froment, "La Préhension dans les paralysies du nerf cubital et le signe du pouce," *Presse médicale*, October 21, 1915.

second phalanx will be seen to be bent and the tip of the thumb will reach to the base of the index finger, forming with it a sort of capital O.

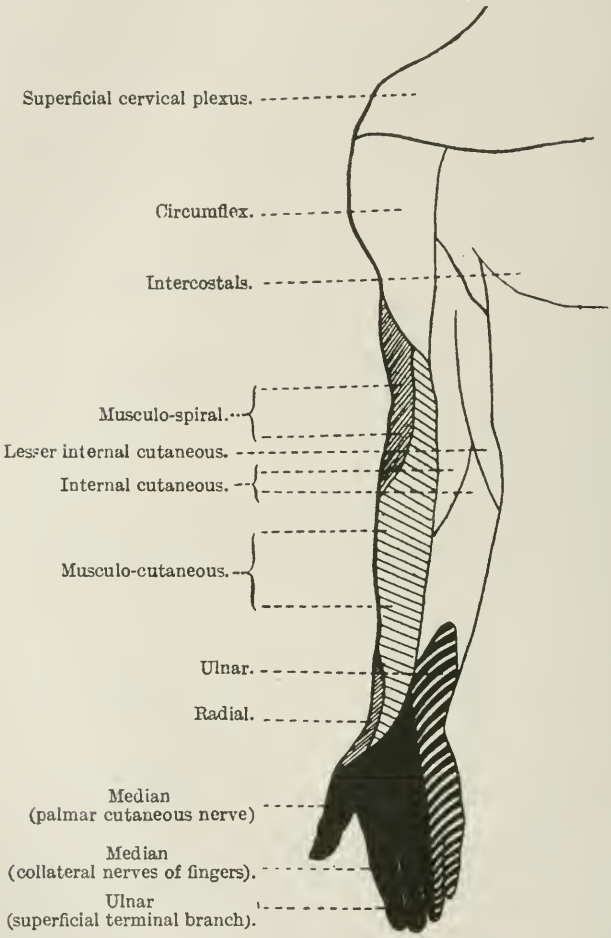


FIG. 38.—Peripheral sensory distribution of the upper extremity (palmar aspect).

So we see that a severe lesion of the ulnar nerve results in marked impairment of movements of the hand. This impairment is less than the impotence that results from paralysis of the median nerve, but

it is quite as important as the impairment of movement that follows injury of the musculo-spiral nerve. The presence of the claw-hand, weakness of grasp through paralysis of the adductor and of the interossei, and flexion of the index from diminished power of extension of its last two phalanges, lead to marked trade incapacity, particularly if it is the right hand that is affected.

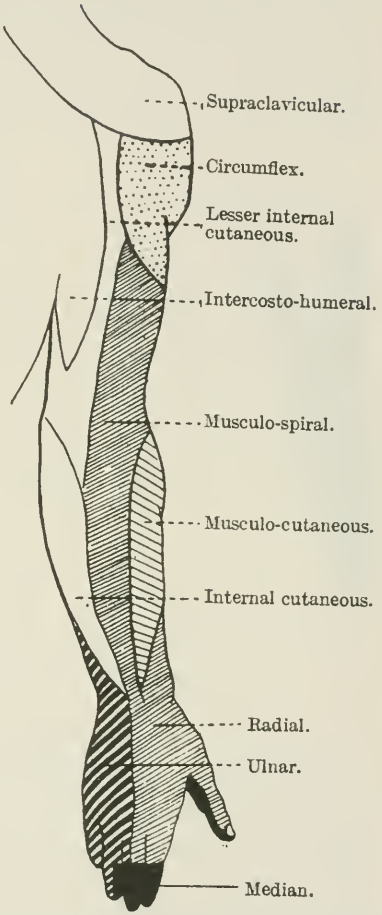


FIG. 39.—*Peripheral sensory distribution of the upper extremity* (dorsal aspect).

Electrical Disturbances.—In case of complete division of the ulnar nerve there is always complete reaction of degeneration in all the paralysed muscles, with loss of faradic excitability, slowness of the contraction, and very marked diminution of galvanic excitability when the wound is of several months' standing.

Reflexes.—The tendon reflexes are not affected by ulnar paralysis. On mechanical percussion of the muscles, *idiomuscular contraction* is slow. The slowness in release of contraction is easy to verify in the hypothenar eminence and the first dorsal interosseous space.

Sensory Changes.—The sensory disturbances are important enough to be considered. We have always been impressed by the intense anæsthesia in the ulnar

area of the hand, even when the wound had not produced total section of the nerve, and sometimes even when the lesion was not serious.

In case of complete section, sensory disturbances are noticed in the ulnar area, as shown in the diagrams (figs. 38 and 39). Complete anæsthesia, however, to all modes of sensibility is only permanent in the little

Fig. 40.

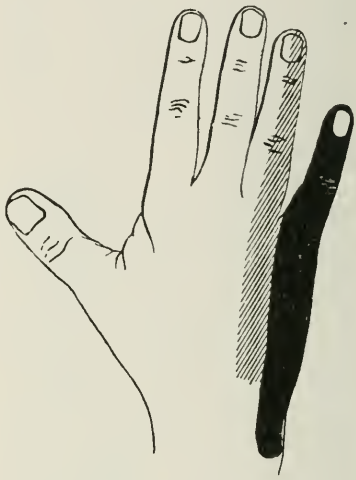


Fig. 41.

Figs. 40 and 41.—Area affected by *sensory changes in grave lesions of the ulnar nerve. Black :* total anæsthesia. *Grey :* hypæsthesia to pricking, anæsthesia to heat and cold.

finger (the whole of which is always insensitive to touch, pricking, deep pressure, heat, and cold) and on the inner margin of the hand ; traces of accidental burns may often be noted in this area (figs. 40 and 41). In the rest of the ulnar area there is only a more or less marked hypæsthesia to a pin-prick, with often complete anæsthesia to heat and cold.

Sensibility of bone and sense of position in the little finger are lost. Pain on deep pressure along the course of the ulnar nerve is not felt when the nerve has been completely divided.

The reason why sensory disturbances are so great is that the sensibility of both dorsal and palmar aspects of the hand in that area depends on the ulnar nerve.

Vasomotor, Secretory, Caloric, and Trophic Changes are often perceptible.

The skin is always cold and dry in the little finger and hypothenar eminence. Sometimes a purplish discoloration is noticed, with thickening of the skin and deformity of the nail of the little finger, which is indicative of an accompanying vascular lesion. The patient should be asked if he has had much hæmorrhage, a ligature, etc.; the ulnar and even the radial pulse as well as the blood-pressure will be affected.

We have already mentioned the muscular atrophy of the interosseous spaces, hypothenar eminence, and the adductor of the thumb. In consequence of this atrophy the flexor tendons project into the palm and the tendons of the extensors stand out on the back of the hand.

When atrophy of the flexor carpi ulnaris is pronounced, a slight displacement of the hand to the radial side may be noticed; palpation reveals decrease in size of the epitrochlear muscles in comparison with the same muscles on the sound side.

RECOVERY

Sensation is usually restored before movement is regained. The latter begins in the flexor carpi ulnaris; then there is improvement in the flexor profundus digitorum, which may make the clawing more marked. The return of movement is very slow in the interossei, hypothenar muscles, and in the adductor pollicis.

Lesions of the Ulnar Nerve in the Forearm

When there is severe lesion of the ulnar nerve *in the forearm or wrist, below the spot where the fibres of the flexor carpi ulnaris and the flexor profundus*

emerge, clawing is greatly accentuated in the little and ring fingers, and often in the middle finger also, but less commonly, and to a minor degree, in the index finger. Hyperextension of the first phalanx and flexion of the other phalanges are exaggerated.

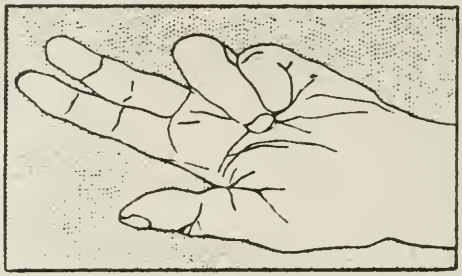


FIG. 42.—*Ulnar clawing* (palmar aspect).

Retraction of the tendons occurs, which makes this clawing quite irreducible.

In addition to the tonicity of the flexor profundus and sublimis other factors must be incriminated, such as muscular contraction, retraction of tendons, surface

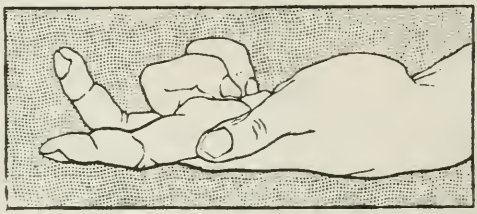


FIG. 43.—*Ulnar clawing* (lateral aspect).

changes in the phalangeal joints, and muscular or vascular lesions which are not at once perceptible (figs. 42–45).

Usually clawing follows immediately on injury; less commonly it develops a few hours or a few days after. Other patients—but more frequently cases of partial lesion—state that at first the last four fingers were clawed, and that later on the index and middle fingers came straight again.

Sometimes for a few minutes after injury the fingers become flexed spasmodically and then settle down into the " claw-hand " position.

In many instances of marked clawing, *spasmodic clonic-like movements* of the middle but not so often

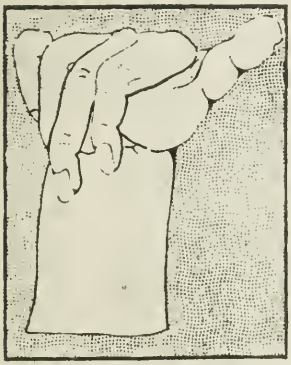


FIG. 44.—*Complete ulnar clawing*, involving all the fingers. Lesion of ulna in forearm.

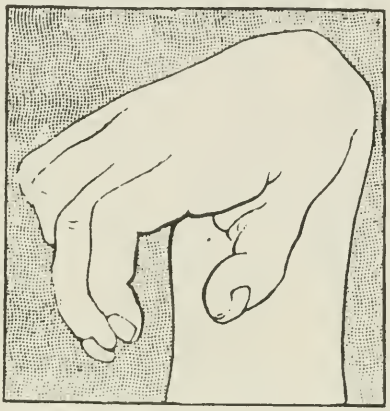


FIG. 45.—Complete ulnar clawing, involving all the fingers. Lesion of ulnar nerve in forearm (dorsal aspect).

of the index fingers may be noticed when the patient puts his thumb in opposition to his fingers.

The other paralytic, sensory, and trophic disturbances, etc., are identical with those seen when the nerve has been completely divided by injury in the arm.

II. PARTIAL PARALYSIS

Lesions of partial, incomplete, or dissociated paralysis of the ulnar nerve are very frequent and interesting, particularly in cases of wounds in the axilla or arm. The commonest type is paralysis of the interossei and hypothenar muscles, with simple paresis of the flexor profundus and flexor carpi ulnaris. In this type of case we have come across limp clawing of the fourth and fifth fingers, pronounced when the hand is at rest, but almost entirely reducible. In some

partial paralyses *total absence of clawing* may be noted; the muscles in the hypothenar eminence and the first interosseus are most severely paralysed, the first dorsal interosseous space is much wasted, and the little finger permanently abducted.

Clawing may involve the last four fingers, though it is most difficult to overcome in the fourth and fifth fingers. Often there is an associated lesion of the median nerve, and in such cases the thumb may be deformed [1] by flexion of the second with extension or hyperextension of the proximal phalanx.

The site of the wound and the distribution of the nerve-fibres should explain the frequency of partial paralysis of the ulnar and the predominance of motor changes in the interossei and hypothenar muscles. Pierre Marie, Henry Meige, and Gosset's recent electrical experiments point to localisation of the nerve-fibres supplying the flexors of the ring and little fingers in the postero-external area, and localise the fibres of the muscles of the flexor carpi ulnaris on the internal side.

It is quite logical to assume that the small muscles of the hand occupy the anterior and internal ulnar area.

On the other hand, M. and Mme. Dejerine and J. Mouzon [2] locate them as follows :—

Internally : the cutaneous branches superficially, branches of the hypothenar and interossei muscles : the outer interossei being the most external.

Behind : the nerves of the adductor pollicis and of the flexor brevis, and, *externally*, the nerves of the flexor carpi ulnaris and the flexor profundus.

Sensory Changes.—Objective disturbances of sensation, though very common, are less marked than in total paralysis, and take the form of hypæsthesia.

[1] Jeanne (de Rouen), *Bulletins et Mémoires de la Société de Chirurgie de Paris*, March 23, 1915, p. 703.

[2] M. and Mme. Dejerine and J. Mouzon, "Le Diagnostic de l'interruption complète des gros troncs nerveux des membres," *Presse médicale*, March 2, 1916.

Pressure on the course of the nerve-trunk and on the hypothenar muscles usually causes pain.

The intense pain invariably felt at the time of injury may persist and may sometimes become a very acute neuralgic pain all along the course of the nerve to the distal end of the little finger, and may be affected by changes of temperature, jarring, and massage.

Causalgia of the ulnar nerve alone we have never seen; it usually occurs in association with a painful type of lesion of the median nerve.

Electrical Changes vary in the different muscles. They are generally most pronounced in the muscles of the hypothenar eminence and last interossei.

DIAGNOSIS

A lesion of this nerve, whatever form it may assume, can always be detected by clinical examination.

The surest signs of lesions of the ulnar are the constant atrophy of the first dorsal interosseous space, the more or less pronounced clawing, the permanent abduction of the little finger, and the inability of the middle finger to make lateral movements.

Ulnar paralysis cannot be easily mistaken if muscular, tendinous, and cicatricial lesions are first eliminated. These lesions, by restricting the play of the flexors, may simulate clawing and mislead the observer.

There is, however, one deformity of the hand which might complicate the diagnosis: the clawing seen in *ischæmic myositis of the flexors*, or *Volkmann's contracture*. But in a case of this kind palpation of the fleshy parts of the muscles will reveal their woody consistency, and the possibility of straightening this clawing when the wrist is forcibly flexed; and the previous history of the case (pressure by a plaster apparatus or arrest of abundant hæmorrhage by a tourniquet) or the presence of vascular disturbance, will lead to a correct diagnosis.

Signs of Recovery.—Complete recovery from a lesion of the ulnar nerve may be shown by the following tests :—

The patient should be instructed to lay the palm of his hand flat on the table with the fingers apart. He should be asked to draw the middle finger inwards and outwards, then told to scratch the top of the table with the nail of his little finger without moving his wrist (Pitres).

From what we have already said about the motor changes it will be clear that the correct performance of these movements indicates complete recovery of the ulnar nerve.

CHAPTER V

ASSOCIATED LESIONS OF THE MEDIAN AND ULNAR NERVES

Concomitant lesions of the median and ulnar nerves are of frequent occurrence. They account for the great variations in appearance of the hand and fingers, and therefore require a full description.

The appearances are the same whether the injury to the nerves is in the arm or in the axilla. Very often there is an associated lesion of the brachial or axillary artery, accompanied by vasomotor, secretory, and trophic changes.

Two clinical types can be distinguished :—

I.—Total paralysis.
II.—Partial paralysis.

I.—Total Paralysis

When the two nerves have been divided or crushed by a missile which has wounded the arm on its inner aspect, or when the nerves have been badly damaged in the axilla at the lower part of the brachial plexus, the hand is quite typical in appearance.

Appearance of the Hand.—The wrist is slightly hyperextended on the forearm, the hand inclines to the radial side, the thumb is in abduction and on a level with the other fingers, which are slightly separated. Their first phalanx is moderately extended on the metacarpus, with slight passive flexion of the last two phalanges without clawing. In very rare

cases the last two fingers may be bent back and show a slight but irreducible clawing.

The hand is wasted ; the fleshy parts of the thenar and hypothenar eminences are flattened, and the first dorsal interosseous space is very hollow.

Motor Changes.—The manner in which the patient tries to flex his wrist and fingers is even more characteristic than the appearance of the hand.

When he is asked to bend his wrist, hyperextension of the hand is exaggerated, and it tilts over to the radial side ; then sudden relaxation follows, the patient lets the extensors of the wrist go, and his hand makes a slight passive movement of flexion. Active contraction of the abductor longus pollicis assists in carrying out this movement of flexion.

But if the patient is told to flex his fingers, the pretence of active movement is even more marked : first, he forcibly extends his hand, the proximal phalanges being hyperextended ; then the extensor muscles of the fingers suddenly relax, and the last two phalanges partially flex, but not in reality. Sicard and Gastaud, Claude, René Dumas and Porak have all investigated the mechanism of this movement, and Sicard and Gastaud have described this movement of alternate contraction and relaxation of the extensors by the term " strumming sign."

But if hyperextension of the first phalanges is prevented by opposing the movement of the extensors, flexion of the fingers is impossible.

Movements of flexion, opposition, and adduction of the thumb are lost ; the patient tries and succeeds in forcibly contracting the extensors and his abductor longus pollicis in order that their relaxation may produce the desired movements of flexion or opposition. But if, while palpating the thenar eminence, the observer makes the base of the thumb immobile, there is no flexion of the second phalanx, and all attempt at opposition is fruitless.

Movements are abolished in the hypothenar, interosseous, and lumbrical muscles, and the patient cannot

replace them in any way. Energetic contraction of the extensors merely produces the faintest suggestion of slight abduction of the fingers.

The elbow must be abducted before pronation of the forearm is possible.

The power of resistance of the biceps is often diminished, as the wound may have caused destruction of the fleshy parts or lesion of the motor nerve of this muscle.

Amyotrophy.—We have already dwelt on the wasting of the muscles of the hand. In addition, there may also be considerable atrophy of the epitrochlear muscles, the flattening of which is in sharp contrast with the prominence of the supinator longus. Though the other nerves may be sound, the muscles of all the segments of the upper extremity show a general decrease in size.

Electrical Changes are the same as those seen in severe lesions of the nerves.

Sensory Changes.—The sensory changes are exaggerated. There is total anæsthesia over all the ulnar area and in the two terminal phalanges of the middle and index fingers. Sometimes this anæsthesia extends over almost all the median area. Most commonly the thumb, outer half of the palm of the hand, and sometimes the palmar aspect of the proximal phalanges of the second and third fingers remain sensitive to a very deep prick. This sensibility is accounted for by the probable assistance given by the fibres of the musculo-spiral and musculo-cutaneous nerves.

Sense of position, deep sensibility, and sensibility of bone are lost in these areas.

The stereognostic sense is lacking by reason of the motor and sensory changes.

Spontaneous pain is absent as well as pain on pressure of the nerve-trunks, which only develop with the onset of recovery.

Vasomotor, Secretory, and Caloric Changes are nearly always present. The hand is somewhat purple

and cold. The skin is dry, especially on the hypothenar eminence; sweating is confined to the back of the hand. But when there are pronounced vasomotor changes, with signs of œdema of the fingers, glossy, cyanosed, or purplish skin, and trophic changes consisting in ulceration and deformity of the nails, an associated vascular lesion should be suspected, because, on account of the close proximity of nerves and arteries, this lesion is very common.

II.—Partial Paralysis

Partial paralyses of the median and ulnar nerves assume very varied clinical types: the accompanying diagrams illustrate this more clearly than any discursive description could do.

The great variety of deformities of the hand and fingers, as the result of affection of both median and ulnar nerves, can only be accounted for by the inequality of their several lesions. Appearances differ according as to whether the graver lesion is in the median or the ulnar, whether this or that fasciculus of each nerve is affected, according to the frequency of anastomosis with the adjacent nerves, and finally, we are inclined to think, according to the more or less inflammatory nature of the lesion.

The phenomena noticed in partial lesions of the median and ulnar nerves are the best illustration of the specialisation of the motor nerve filaments in the interior of a nerve.

The accompanying drawings, made from nature, give a very good idea of the different varieties of deformity.

We shall deal with five chief clinical types:—

First type.—The most common type is that in which motor disturbances predominate in the small muscles of the hand and in the flexors of the fingers, the flexors of the wrist being comparatively unaffected. Flexion of the wrist alone is possible.

The *position* of the hand and its *loss of power* closely

resemble the conditions described above in cases of complete division of the nerves.

The *sensory disturbances* are confined more especially to the ulnar area, and usually result in hypæsthesia rather than anæsthesia.

Spontaneous pain along the course of the ulnar nerve

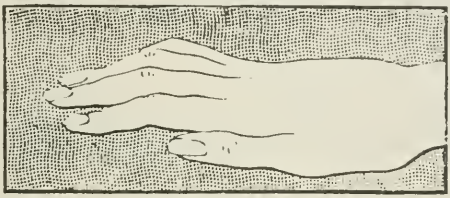


FIG. 46.—Paralysis, involving both *median and ulnar nerves* (first type).

is not uncommon. Palpation of the nerve-trunks is always painful.

Electro-diagnosis shows reaction of degeneration

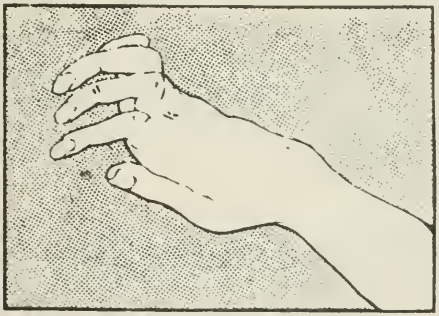


FIG 47.—Associated lesion of *median and ulnar nerves* from wound of axilla. Paralysis of small muscles of hand with paresis of flexors (second type).

unevenly distributed; faradic contractility is not abolished in all the muscles.

In this type of case there is comparatively marked *atrophy of the muscles above the lesion*, with pain on pressure of the nerve-trunks above the injury.

Second type.—The hand has a most singular appear-

ance when paralysis chiefly affects the interossei, thenar and hypothenar muscles, and when there is paresis of the superficial, with more marked paresis of the deep, flexors. Hyperextension of the proximal

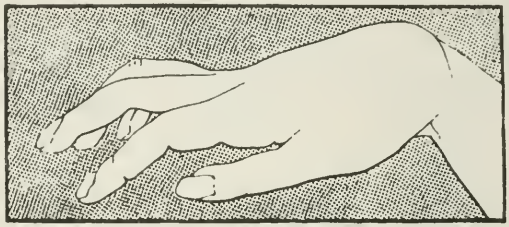


FIG. 48.—Associated lesion of *median and ulnar nerves* from wound in the cervical region (second type). Motor symptoms are most marked in the small muscles of the hand.

phalanges of the last four fingers is so great as to cause a subluxation of the heads of the metacarpal bones which project into the palm of the hand at the base of the fingers (fig. 47).

Flexion of the second phalanx is good, that of the

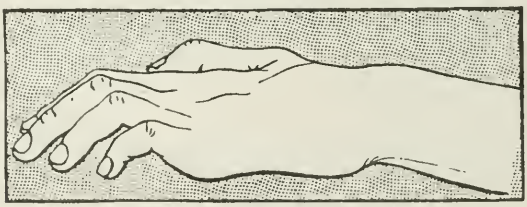


FIG. 49.—Associated paralysis of *median and ulnar nerves* from wound of the arm (third type). Motor symptoms are most marked in the small muscles and in the deep flexors.

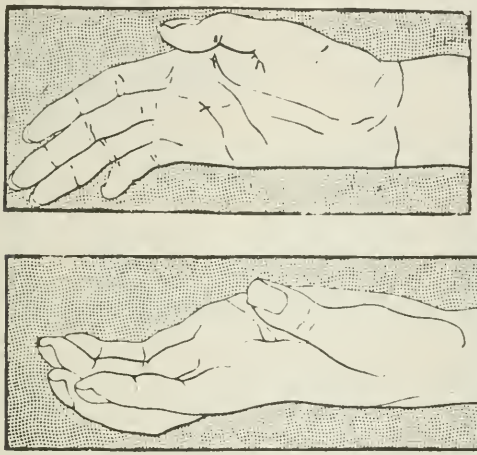
terminal phalanx is less so. The flexor tendons stand out in the palm of the hand, which is emaciated.

Looked at from the dorsal aspect, the hand resembles the back of a fork. Sometimes, in addition, disturbance of function of the *vasomotor fibres* accompanies this type of hand. In one of our cases the ring and

little fingers were always hot, and the index and middle fingers cold.

There is also a tendency to *ankylosis* of the joints, which perhaps explains the subluxation of the heads of the metacarpals and the stiffness of all the knuckles.

Sometimes, in analogous cases, the same muscles are affected to a less degree, and then there is a slight movement of abduction and adduction, but flexion of the first phalanges on the metacarpus is always impos-



FIGS. 50 and 51.—Associated paralysis of the *median and ulnar nerves* from wound of the axilla (fourth type). The flexor sublimis is much affected.

sible. There is the same "fork back" appearance (fig. 48).

Third type.—The small muscles of the hand are the ones most affected, but hyperextension of the dorsal phalanx is less pronounced (fig. 49). The patient can close his fist; the fingers bend a little at the metacarpo-phalangeal joint, fairly well at the first interphalangeal joint, and not at all at the junction of the second and terminal phalanges. The second phalanx of the thumb is in permanent flexion, and shows the typical deformity described by Jeanne (of Rouen). In spite of the way in which the heads of the metacarpal bones project into the hand, this clawing is very easily overcome: the "fork back" is less pronounced.

Fourth type.—The superficial flexor is very much

affected in this type; the fibres of the deep flexors supplied by the ulnar nerve are not so much involved, and this accounts for the faint attempt at flexion of the fourth and fifth fingers and the slight clawing (figs. 50 and 51).

Though paralysed, the interossei react fairly well to the faradic current.

In the early stage there was paresis of the muscles

FIG. 52.—Partial paralysis of *median and ulnar nerves* (fifth type). Associated vascular lesion.

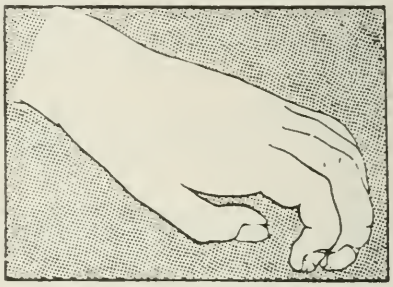


FIG. 53.—Partial paralysis of *median and ulnar nerves* (plexus lesion) (fifth type). Associated vascular lesion.

supplied by the musculo-spiral nerve, followed by complete recovery.

Fifth type.—In this type, too, the interossei are deeply implicated; the flexor longus pollicis is normal; the flexors of the fingers partially contract; the thenar muscles, which at first were paralysed, are beginning to recover.

In the two cases which we illustrate there was an associated vascular lesion, fairly well compensated for in most respects, and irritation of the secretory fibres which led to excessive sweating in the median area (figs. 52–54).

Finally, in all these different types the amount of paralysis of the interossei and flexors should be noted on the one hand; and, on the other, the strength of the extensors, associated vascular lesions, anastomoses of the nerve-trunks, and, finally, articular lesions should be taken into consideration.

Articular lesions no doubt depend on individual susceptibility to disease; but we are inclined to think that the lesion is related to those arthropathies which set in early in some wounded men and result in great deformity.

The retraction of the flexor tendons varies in different individuals, though the site of lesion in the nerve

FIG. 54.—Partial paralysis of *median and ulnar nerves* from injury to the brachial plexus (fifth type). Concomitant vascular lesion and retraction of tendons.

may be identical. Is this due to predisposition on the part of the patient or to degeneration of muscle? Or is there any established equilibrium between the flexor sublimis, the flexor profundus, and the interossei, which varies with the individual, and the upset of which presents different features according to the type of the lesion?

In these partial palsies it may be as well also to take into consideration the progressive, though unequal, return of movement and tone to some of the muscles which, as they recover, cause twitchings and alter the equilibrium of the articular surfaces, but, when inflammation of the soft parts supervenes, the lesion is established, and ankylosis and retraction set in. Only vigorous and prolonged treatment avail then to effect any change in the conditions.

EXPLANATION OF PLATE IV.

A. *Persistent ulceration of the tip of the fingers, involving the nails and ending in retractile cicatrices. It was a case in which there had been complete division of the median and ulnar nerves complicated by much damage to the brachial artery.*

B. *Infiltrated and succulent hand in a case of lesion of the axillary artery, with complete division of the ulnar and contusion of the median nerve.*

Vasomotor (discoloration and œdema), thermic (coldness), and secretory (absence of sweating) symptoms are most marked in the last three fingers.

C. *Serious injury to the ulnar artery on the right side at the lower third of the forearm.*

Irreducible retraction of the interossei, probably of ischæmic origin, and deformity of the terminal phalanges of the fingers resembling some forms of chronic rheumatism.

D. *Associated lesion of ulnar and median nerves; wound of brachial artery. Profile of hand.*

Considerable atrophy of thenar, hypothenar, and interosseous muscles.

"Ape-hand" clawing of all the fingers, but more pronounced in the ring and little fingers; marked projection of the heads of the metacarpal bones into the palm of the hand. Nevertheless the clawing is passively reducible.

A

B

C

D

PLATE IV.

CHAPTER VI

THE MUSCULO-CUTANEOUS NERVE

This nerve is rarely affected alone; more commonly it suffers when the brachial plexus is injured. We have also seen paralysis of this nerve complicated by musculo-spiral paralysis in the arm and by dissociated paralysis of the median nerve. The nerve is then nearly always injured at the spot where it pierces the coraco-brachialis.

ANATOMY

The musculo-cutaneous nerve leaves the external root of the median nerve in the axilla. Its fibres are derived from the fifth and sixth cervical nerves. At its exit from the axilla it perforates the coraco-brachialis muscle, passes obliquely between the biceps in front and brachialis anticus behind to the outer side of the arm, and a little above the elbow becomes subcutaneous by perforating the deep fascia on the outer side of the tendon of the biceps. It then divides opposite the elbow joint into an anterior and posterior branch and supplies the integument of the outer half of the forearm (figs. 16, 38, and 39).

In its course through the arm the musculo-cutaneous nerve supplies muscular branches to the *coraco-brachialis*, *biceps*, and *brachialis anticus*.

The branch to the *coraco-brachialis* is given off from the nerve close to its origin under the lower border of the pectoralis major. The filament to the biceps is supplied after the nerve has pierced the coraco-brachialis, and it divides into two branches for the two heads of the biceps.

The branch to the *brachialis anticus* is given off a little below that to the biceps.

It is quite possible that the terminal cutaneous branches of the musculo-cutaneous nerve supply the integument of the first dorsal interosseous space as far as the triangle between the thumb and index finger and much of the integument of the thenar eminence, taking the place—as regards sensibility—of the radial and median when these nerves have been injured.

The musculo-cutaneous nerve has also probably something to do with the surprising preservation of superficial sensibility in the area of the external cutaneous branch of the musculo-spiral nerve. Lastly, the important and frequent communications between the musculo-cutaneous and the median nerves along their course in the arm should not be overlooked.

PHYSIOLOGY

The *coraco-brachialis* muscle retains the head of the humerus firmly in the glenoid cavity of the scapula when the arm is lowered, and it thus assists in this movement.

It is difficult to estimate by clinical examination alone an isolated affection of this muscle.

But the same cannot be said about the biceps and the brachialis anticus which flex the forearm on the arm.

The *brachialis anticus* is an independent flexor of the forearm, *i.e.* it produces vigorous flexion of the forearm, without any other movement.

The *biceps* flexes and supinates the forearm. To obtain proof of this functional activity it is only necessary to apply a tetanising faradic current of some intensity to the biceps in a patient suffering from paralysis of the median nerve, the elbow being in slight flexion, the hand in pronation with the tonicity of the pronators *nil*, or much diminished (Babinski). In flexing the forearm the biceps also completely supinates the hand. It is with the assistance of the biceps that patients with musculo-spiral paralysis can supinate their hands. According to Duchenne (of Boulogne) the performance of this movement of supination is due rather to the short than to the long head of the biceps.

CLINICAL EXAMINATION

Motor Changes.—In case of a severe lesion of the musculo-cutaneous nerve from complete division, contusion, or compression of the nerve-trunk by imprisonment in a callus, the muscles of the anterior humeral region are totally paralysed. If the musculo-spiral nerve is intact the patient can flex the forearm on the arm very well, owing to the aid of the supinator longus. But the power of resistance is lessened, and palpation of the muscular parts of the biceps and brachialis anticus shows that these muscles are absolutely flaccid.

In case of concomitant musculo-spiral and musculo-

cutaneous paralysis either in the arm or axilla, active flexion of the forearm becomes quite impossible, because the muscles of the anterior humeral region and the supinator longus are paralysed. The arm hangs down by the side of the body in limp extension ; if the patient's forearm is raised and let go again, the suddenness of the drop depends on how much the tonicity of the pronator radii teres is affected, which is an accessory flexor of the forearm.

Muscular *atrophy* is usually very marked.

Electrical Changes.—Reaction of degeneration is the rule ; quantitative changes are proportionate to the severity of the lesion.

Sensory Changes are sometimes very pronounced in the musculo-cutaneous region ; at others, there is hypæsthesia to pricking, heat, and cold.

When the musculo-cutaneous nerve is affected near its origin without being divided, there is simultaneous paresis of the biceps and brachialis anticus, with *neuralgic* pains in the region of this nerve, which are most marked at the bend of the elbow and in the first dorsal interosseous space, where the pain may become *causalgic*.

Reflexes.—The stylo-radial reflex only shows contraction of the supinator longus.

Idiomuscular contraction, which is exaggerated at first, becomes less, and in the case of the biceps may show some slowness in release of contraction.

Recovery.—Recovery of the powers of movement and sensation proceeds simultaneously, and in a few months' time there is voluntary contraction of the biceps and the sensory symptoms begin to disappear. Huet has repeatedly insisted that the length of a nerve should be taken into consideration in estimating the repair of nervous lesions.

In lesions of the plexus, and particularly in lesions of the upper roots of the brachial plexus, recovery may often be seen to begin in the circumflex nerve, then appear in the musculo-cutaneous, and finally occur in the branch to the supinator longus (Huet).

CHAPTER VII

THE CIRCUMFLEX, INTERNAL CUTANEOUS, AND LESSER INTERNAL CUTANEOUS NERVES

I.—The Circumflex Nerve

As in several hundreds of cases of peripheral nerve lesions we have never come across isolated paralysis of the circumflex nerve, such cases must be rare.

The anatomy of the circumflex nerve, the normal and pathological physiology of the deltoid and teres minor muscles supplied by it, as well as the sensory changes which its injury causes, will be described in connection with paralyses of the brachial plexus. Lesion of the circumflex or of its branches is in fact always associated with paralysis of other collateral or terminal branches of the cervical or brachial plexus.

II.—The Internal Cutaneous Nerve.

Lesions of this exclusively sensory nerve frequently accompany paralysis of the median or ulnar, or lesions of both these nerves (Laignel-Lavastine).

Anatomy and Physiology.—The internal cutaneous nerve arises from the inner cord a little internal to the origin of the ulnar and inner head of the median. It derives its fibres from the eighth cervical and first dorsal nerves.

It passes along the axilla and inner side of the arm in close relation to the artery and the median and ulnar nerves.

It pierces the deep fascia about the middle of the upper arm, and soon divides into two terminal branches—an anterior, which supplies the skin of the anterior and internal aspects of the forearm ; and a

posterior and more slender branch, which supplies the dorsal and internal aspects.

The internal cutaneous nerve distributes two or three filaments to part of the anterior aspect of the arm (figs. 38 and 39).

It frequently communicates with all the other nerves of the upper extremity.

When the nerve has been divided there is a long narrow area of anæsthesia, which is very marked on the inner side of the forearm.

In case of pressure only, numbness over the inner side of the forearm, spreading to the anterior and posterior aspects, is complained of.

The internal cutaneous nerve seems to be able to resist considerable pressure; for in lesions affecting the median and ulnar it is very frequently involved in the fibrous tissue which has formed round the wounded nerves, without any considerable sensory changes taking place in its cutaneous area.

III.—The Lesser Internal Cutaneous Nerve.

The function of this nerve is exclusively sensory, and like the internal cutaneous, it arises from the inner cord of the brachial plexus and receives its fibres from the first dorsal.

It communicates with the lateral cutaneous branch of the second and sometimes of the third intercostal nerve, and supplies the skin of the inner part of the arm (figs. 38 and 39).

When the lesser internal cutaneous nerve is thought to be affected, it is in this area that sensory disturbances should be looked for.

CHAPTER VIII

THE CERVICAL AND BRACHIAL PLEXUSES

Paralyses of the nerves of the brachial plexus are of very common occurrence and very varied in nature, but this is not surprising if the complexity of the large nerve-trunks in the neck and axilla and the many nerve-fibres which are distributed to the muscles from it are borne in mind.

This complexity makes it essential to know the anatomy of this region and the physiology of the muscles supplied by the branches of the plexus. A minute clinical examination of the paralytic conditions on the one hand and the changes in objective sensibility on the other will enable the clinician to localise the situation of the lesion, and to know precisely whether the injury has damaged the cervical roots where they emerge, or after the formation of the primary or secondary trunks of the plexus.

To do this it is essential to have a diagram before one's eyes showing the exact height at which the different motor nerve branches are given off and their proximity to one another. Henry Meige[1] has drawn up several diagrams of this description which not only have been most useful to us, but have also proved an invaluable guide to the surgeon in operations in the region of the cervical and brachial plexuses.

Plexus paralysis results from wounds in the axilla or neck; frequently the projectile has entered the

[1] *Société de Neurologie*, June 29, 1916.

cervical region and come out through the supra- or infraspinous fossa; less commonly the face is the point of entry, and the neck or axilla of exit.

Both from the anatomical and physiological points of view, it will be advisable to describe the cervical plexus immediately after the brachial, in view of the possible variation in the course of the projectile and the proximity of the cervical roots to one another. For though isolated lesions of the cervical are somewhat rare, lesions involving both brachial and cervical plexuses are more frequent.

ANATOMY

The *brachial plexus* is formed by the union of the anterior divisions of the fifth, sixth, seventh, and eighth cervical nerves and of the first dorsal nerve.

It is easy to make a fairly simple plan of the brachial plexus.[1]

The anterior division of the fifth cervical in its descending course unites with that of the sixth cervical to form a common trunk called the *upper primary trunk.*

The anterior divisions of the eighth cervical and of the first dorsal unite at the junction of the posterior and middle thirds of the first rib to form the *lower primary trunk.*

Between the upper and lower trunks the anterior division of the seventh cervical passes horizontally and does not divide; it is called the *middle primary trunk.*

Each of these three primary trunks divides into two branches—an anterior and a posterior—which communicate and unite to form secondary trunks.

The anterior division of the upper primary trunk unites with the anterior division of the middle primary trunk to form the *upper or outer secondary trunk*; the anterior division of the lower primary trunk forms by itself the *lower or inner secondary trunk.*

Finally, the posterior divisions of the three trunks unite to form the *middle secondary trunk*, which is located behind the upper and lower secondary trunks.

It is from the outer end of these secondary trunks that the nerves of the upper extremity, which are regarded as the terminal branches of the brachial plexus, arise.

The outer or upper secondary trunk (upper in the cervical region, and outer in the axillary) gives rise to the *musculo-cutaneous nerve and the outer head of the median.*

The lower or inner secondary trunk (lower in the cervical, inner

[1] A. Soulié, *Les nerfs rachidiens*, in Poirier et Charpy, tome iii. fasc. iii.

in the axillary region) gives rise to the *ulnar, internal cutaneous, and the inner head of the median nerve.*

The middle secondary trunk gives rise to the *musculo-spiral and circumflex nerves.*

At the point where the fifth, sixth, and seventh cervical nerves appear between the scalenus anticus and the scalenus medius, they are situated in front of the anterior divisions of the eighth cervical and of the first dorsal. These two nerves are in close relation to the large arteries at the base of the neck and to the dome of the pleura.

The fusion of the fifth and sixth cervicals into an upper primary trunk occurs on the scalenus medius, external to the outer border of the scalenus anticus. This point, which is of importance in electro-diagnosis, is a little external to the outer border of the sterno-mastoid muscle, 1–2 cm. above the clavicle, and is known as *Erb's point.*

The supraclavicular portion of the brachial plexus is composed of radicular and primary trunks. Below the clavicle in the axillary region are the secondary trunks, and it is here that the nerves of the upper extremity arise.

The axillary artery in its almost entire course lies between the secondary nerve-trunks which separate it from the axillary vein. The brachial plexus is also in close relation at the outer wall of the axilla with the glands draining the lymphatics of the upper extremity.

In our study of the large nerve-trunks of the upper extremity, we described the motor and sensory ramifications of the terminal branches of the brachial plexus.

We must now refer to the collateral branches of the brachial plexus. For convenience sake, they are divided into posterior branches or muscular nerves of the dorsal region of the shoulder, and anterior branches supplying the muscles on the ventral side of the shoulder.

A. *The posterior branches* are *seven in number* :—

1. Those which arise on the posterior aspect of the radicular trunks are (*a*) *the nerve to the levator anguli scapulæ and the rhomboidei* (it is derived from the fourth and fifth cervicals); (*b*) *the nerve to the serratus magnus* (fifth, sixth, and seventh cervicals).

2. Those which arise on the posterior aspect of the plexus (primary and secondary trunks), and consequently are more external than the preceding.

(*a*) *The upper subscapular nerve* (C^5, C^6); (*b*) *the lower subscapular nerve* (C^5, C^6, C^7); (*c*) *the nerve to the teres major* (C^6, C^7); (*d*) *the nerve to the latissimus dorsi* (C^6, C^7, C^8); (*e*) *the circumflex nerve* (C^5, C^6).

The last, on account of its comparatively large size sometimes regarded as the terminal branch of the plexus, arises from the posterior secondary trunk of the brachial plexus in common with the musculo-spiral nerve; it passes downward and outward behind

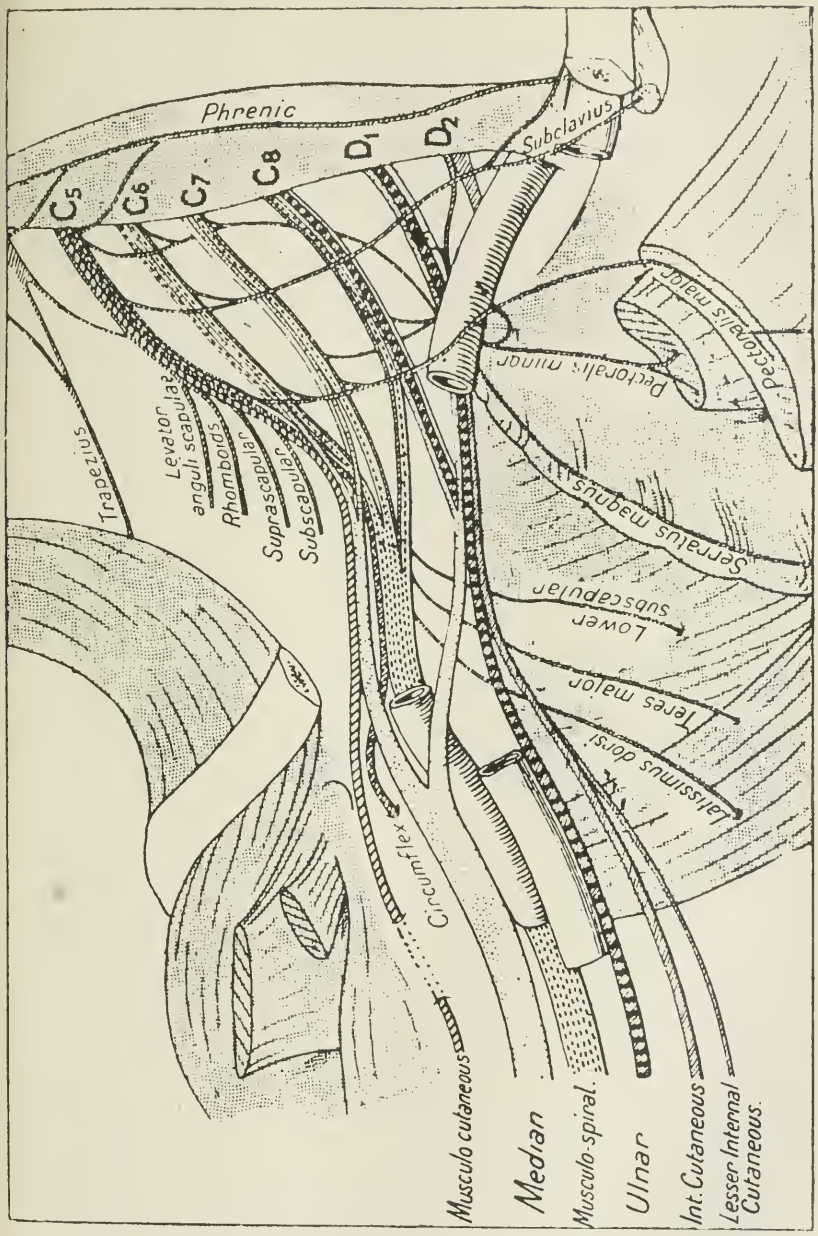


PLATE V.

Brachial plexus. (After Henry Meige.)

the axillary artery a little above the musculo-spiral. In company with the posterior circumflex artery it crosses the surgical neck of the humerus and distributes filaments to the *deltoid muscle*, beneath which it is placed.

In its course it has supplied branches to the axillary artery, to the shoulder-joint, and two more important branches—the *nerve of the teres minor* and the *cutaneous nerve of the shoulder*, which supplies the integument over the deltoid and the posterior and upper half of the arm.

B. *The anterior branches* of the scapular girdle are *four in number*:—

1. *The suprascapular*, which arises from the upper primary trunk (C^4, C^5), winds round the upper part of the plexus and enters the supraspinous fossa. It supplies branches to the *scalenus medius*, but more particularly muscular branches to the *supra- and infraspinatus*, osseous branches for the scapula, and articular filaments to the shoulder-joint.

The other three branches, called anterior thoracic nerves, arise from the anterior aspect of the brachial plexus.

2. *Nerve to the subclavius* (C^5, C^6).

3. *External anterior thoracic nerve, or nerve to the pectoralis major*, arises from the upper secondary trunk of the brachial plexus (C^5, C^6, C^7); it passes in front of the axillary artery, and is distributed to the under surface of the pectoralis major.

4. *Internal anterior thoracic nerve, or nerve to the pectoralis minor*, arises from the lower secondary trunk (C^7, C^8).

Before concluding the brachial plexus we will enumerate the roots from which the terminal branches of the brachial plexus arise:—

The *musculo-cutaneous* is derived from the fifth, sixth, and seventh cervical roots.

The *median* is derived from the sixth, seventh, and eighth cervical and first dorsal.

The *musculo-spiral* is derived from the sixth, seventh, and eighth cervical.

The *ulnar* is derived from the seventh and eighth cervical and first dorsal.

The *internal cutaneous* is derived from the eighth cervical and first dorsal.

The *lesser internal cutaneous* is derived from the first and second dorsal.

The *cervical plexus* is formed by the anterior divisions of the four upper cervical nerves. Each of these anterior divisions at the transverse process of the corresponding vertebra divides into two branches—ascending and descending—which unite with similar twigs from the adjacent branch to form loops, called *cervical loops*.

The three loops formed by the first four cervical nerves form the *cervical plexus*.

The nerves of the plexus spring direct either from the anterior cervical branches or from the communicating loops uniting these branches. For convenience of description they are divided into superficial or cutaneous, named collectively *the superficial cervical plexus*; and into deep or muscular, called the *deep cervical plexus.*

There are four branches of the superficial cervical plexus, all sensory in function :—

1. *The mastoid branch* (C^1, C^2, C^3) divides near the mastoid process into an anterior and posterior branch, and supplies the skin of the lateral occipital region.
2. *The auricular branch* (C^1, C^2, C^3) also divides into two branches, anterior and posterior, which supply the skin in the parotid region.
3. *The transverse cervical branch* (C^2, C^3) divides into an ascending and descending branch, and is distributed to the skin of the antero-lateral aspect of the neck.
4. *The supraclavicular branch* (C^3, C^4), comprising sternal, clavicular, and acromial branches, supplies the skin of the shoulder.

The *deep cervical plexus* consists of muscular and communicating branches. The former are the more important for our purpose.

For convenience sake they are usually divided into internal, external, and descending branches.

There are three internal branches :—

1. *The nerve to the rectus capitis anticus minor* (C^1).
2. *The nerve to the rectus capitis anticus major* (C^1).
3. *The nerves to the longus colli* (C^2, C^3, C^4).

There are six external branches :—

1. *The nerve to the rectus capitis lateralis* (C^1).
2. *The nerve to the sterno-mastoid* (C^2, C^3).
3. *The nerves to the scalenus muscles* (C^3, C^4).
4. *The nerve to the trapezius* (C^3).
5. *The nerve to the levator anguli scapulæ* (C^3, C^4).
6. *The nerve to the rhomboideus* (C^3, C^4).

We should not forget that the trapezius and sterno-cleido-mastoid muscles are supplied in addition by the spinal accessory (eleventh cranial nerve), and that the levator anguli scapulæ and the rhomboid also receive filaments from the brachial plexus.

The descending branches of the deep cervical plexus are two in number :—

1. *The internal descending branch* (C^2, C^3) communicates with the descending branch of the hypoglossus to form a loop (ansa hypoglossi), from the convexity of which branches pass to supply the sterno-hyoid, the sterno-thyroid, and the anterior and posterior bellies of the omo-hyoid.
2. *The phrenic nerve* (C^3, C^4, C^5), for the diaphragm.

PHYSIOLOGY OF THE MUSCLES OF THE SHOULDER

The trapezius.—Total contraction of this muscle lifts the whole scapula, whilst its vertebral border approaches the middle line, and the point of the shoulder is drawn from before backwards and from without inwards; the head is then thrown back and turned to the opposite side.[1]

In clinical medicine, the strength of the trapezius can be tried by asking the patient to shrug his shoulders hard, throwing them back and bringing the two shoulder blades together. The observer should resist this movement by pressing his hands on the patient's shoulders; it is then possible to realise the extent to which the trapezius has lost power.

Normally, in a subject who lets his arms hang down naturally by the sides of the body, the vertebral border of the scapula is nearly parallel with the spinal column, and generally about 5–6 cm. from the middle line.

When the trapezius is paralysed, atrophy may only affect the lower part of the muscle, where it is attached to the inner half of the spine of the scapula and acromion. Then the vertebral border of the bone deviates from the middle line—the more marked the atrophy, the greater the deviation: at the same time the shoulder is lower than on the sound side: the shoulder is tilted forward and the chest becomes hollow.

If atrophy involves all the fibres of the trapezius which are connected with the scapula—spine and acromion—the shoulder drops and the scapula is tilted so that its inner angle deviates from the middle line and approximates to the shoulder; whilst its outer angle drops and its lower angle rises and approximates to the middle line, projecting under the skin.

If a patient with paralysis of the lower part of the trapezius is asked to bring his shoulder-blades together vigorously, the action of the rhomboid is brought into play, elevating and retracting the scapula: it forms a strong oblique cord running from below upwards and from without inwards on its route from the lower angle of the scapula to the cervico-dorsal column. The clavicular portion of the trapezius is supplied both by the cervical plexus and the spinal accessory: it is chiefly respiratory and not much of an elevator. If the whole of the trapezius is paralysed, the shoulder seems to slip away from the thorax, much to the discomfort of the patient.

The *rhomboid*, as we have seen, pulls the scapula upwards towards the middle line, approximating its inferior angle to the vertebral column, while the internal angle is drawn away from it. The trapezius, acting in conjunction with the rhomboid, directly lifts the shoulder; if it is raised energetically the rhomboid chiefly

[1] Duchenne (of Boulogne), *Physiologie des mouvements*, 1867.

comes into play. If this muscle is paralysed and atrophied at the same time as the trapezius, the groove between the spinal column and the scapula becomes deeper. The rhomboid steadies the vertebral border of the scapula, and paralysis of it makes it difficult to draw the arm backwards and outwards.

It is desirable to be able to judge of changes in position and strength of resistance, for, in spite of the loss of function of the rhomboid and of the trapezius, the patient can bring his shoulder-blades together with force by means of the upper fibres of the latissimus dorsi.

The *levator anguli scapulæ*, which passes from the superior angle of the scapula to the transverse processes of the first four or five cervical vertebræ, raises and rotates the superior angle of the scapula inward, drawing the point of the shoulder with it.

The serratus magnus.—Contraction of all the fibres of this muscle results in carrying the scapula forwards, outwards, and upwards; it is an important inspiratory muscle, but in supporting weights it does not contract.

It contracts vigorously, in conjunction with the pectoralis major in the action of pushing forward with the point of the shoulder.

Its action is closely associated with that of the deltoid, and it assists it in raising the arm.

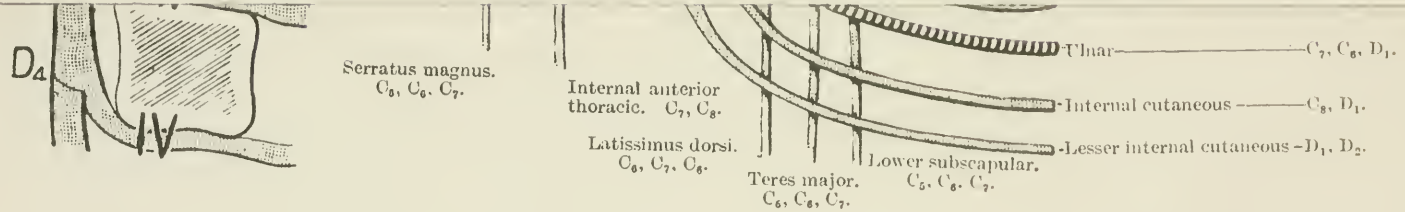
When the serratus magnus is paralysed, the position of the scapula, when at rest, is not abnormal, as it is held close to the wall of the thorax by the trapezius and rhomboid. But if these muscles are paralysed too, the scapula drops completely, its lower angle moves towards the middle line, and its axillary border becomes almost horizontal.

In order to demonstrate paralysis of the serratus magnus, ask the patient to bring his arm forward horizontally. The result will be a very characteristic deformity: the anterior aspect of the scapula leaves the thorax and its vertebral border stands out prominently from the middle line under the skin. The patient is not so well able to lift the arm as on the sound side, for, as the scapula is no longer fixed, the deltoid cannot work properly.

If he is told to raise his arms at the side instead of in front, there is the same difficulty in attaining the horizontal, but the deformity of which we have spoken will be less apparent.

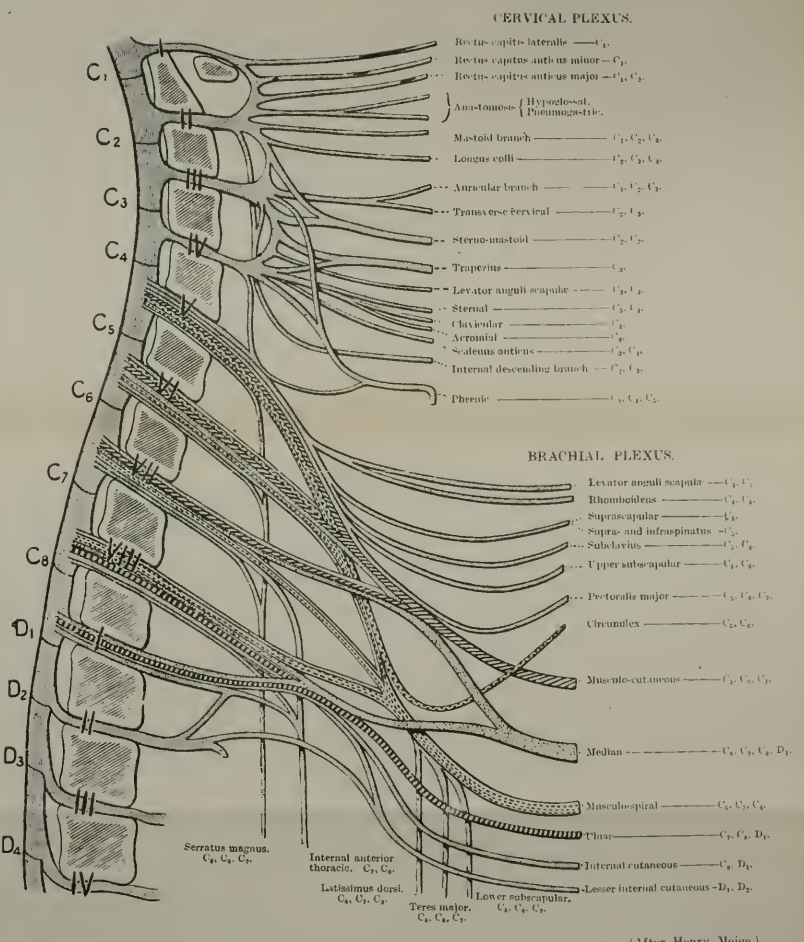
The *deltoid* raises the arm directly from the side to a right angle with the trunk, and in doing so the vertebral border of the scapula stands out from the wall of the thorax. But if it is desired to raise the arm completely, the scapula must turn so that its external angle is raised. After the deltoid has raised the arm to a right angle with the trunk, the serratus magnus with the middle part of the trapezius completes the movement.

Whenever the arm is vigorously raised all the fibres of the deltoid



(After Henry Meige.)

PLATE VI.



(After Henry Meige.)

PLATE VI.

contract. But in some positions a few of the fibres must relax; thus, whenever the arm is drawn forwards or inwards the posterior fibres relax.

Each of the fasciculi of the deltoid should be examined separately, both from the clinical and electrical points of view.

The *supraspinatus muscle* lifts the humerus and draws it forwards and outwards. Though much weaker than the deltoid, it is strong enough to raise the arm when the deltoid is completely atrophied.

The *infraspinatus and the teres minor* are one and the same muscle, physiologically, and rotate the head of the humerus outwards.

The *subscapularis*, on the contrary, rotates the arm inwards.

These three last-named muscles assist in the movements of supination and pronation, as we have already said in studying the lesions of the musculo-spiral and median nerves.

The *pectoralis major* when fully contracted adducts and simultaneously draws the arm forward.

In studying the action of this muscle we should distinguish between the upper portion, *i.e.* the clavicular and upper sternal digitations, and the lower portion, *i.e.* the lower sternal digitations, the costal fasciculi, and the fasciculus attached to the abdominal aponeurosis.

The upper portion in contracting draws the point of the shoulder upwards and forwards. If the arm is raised to the vertical, contraction of this portion of the muscle draws the shoulder forwards and towards the middle line and depresses it to the horizontal.

The lower portion also depresses the upper extremity to the horizontal and below it.

To test the power of resistance of the pectoralis major, ask the patient to keep his elbow firmly flexed against his trunk with the shoulder depressed; he should then oppose any attempt at forcible abduction of the limb. The observer should meanwhile note the strength of the movement, and carefully palpate the muscle to judge of its power of contraction in comparison with the sound side.

The *latissimus dorsi* muscle depresses the shoulder, draws the arm inwards, rotates it backwards, and brings the scapula to the middle line.

When the two latissimi dorsi contract simultaneously, the shoulders become less prominent and are carried obliquely backwards and inwards. At the same time there is strong extension of the dorsal part of the trunk. In short, these two muscles contract in the attitude known as "shouldering arms." In testing the power of resistance of the latissimus dorsi, the patient should be told to place his affected hand behind his back on the buttock of the sound side, and in this position to resist the observer's attempts to draw the arm forwards.

The *teres major* raises the shoulder, brings the inner aspect of the

arm and the axillary border of the scapula together, and draws the humerus backwards : its action in depressing the arm is weak.

The *sterno-cleido-mastoid*, as it contracts, moves the head in three ways :—

1. It bends it on the spinal column.
2. It draws it to the shoulder of the same side.
3. It rotates the head so as to carry the chin to the opposite side.

To test the power of resistance of the left sterno-mastoid, for instance, the patient should be asked to bend his head slightly, and to turn it to the right, and then asked to resist the observer's attempt to draw it to the left by taking hold of his chin.

When the muscle is paralysed, its power of resistance is diminished and it no longer stands out under the skin as it does normally.

The *scalenus anticus* is an inspiratory muscle ; it also serves to turn the head to its own side.

Simultaneous bilateral contraction of the scaleni muscles makes the cervical column very rigid, by immobilising the vertebræ into which they are inserted.

Too much importance must not be attached to the clinical examination of the muscles of the infrahyoid region.

Electrical examination will, however, soon show whether they are affected.

The muscles of the anterior vertebral region supplied by the first cervical roots (rectus capitis anticus major, rectus capitis anticus minor, etc.) are of minor importance, and their clinical and electrical examination is difficult or impossible.

Various Clinical Types of Plexus Paralysis

I. LOWER PLEXUS PARALYSIS

This is limited to the common trunk of the *median and ulnar nerves*.

We have already given a brief description of the different kinds of deformity produced by total and partial paralysis of these two nerves.

The symptoms are the same when the axilla is the site of the lesion.

At the onset the lesion of the plexus has probably been more extensive, involving the musculo-spiral or, less commonly, both the musculo-spiral and the musculo-cutaneous nerves ; but when these nerves have recovered their function, the only sequela which persists is affection of the median and ulnar nerves.

II. UPPER PLEXUS PARALYSIS (C^5, C^6)

Paralysis of the upper roots of the brachial plexus, due to lesion of the fifth and sixth cervical roots, is well defined and fairly common (Erb-Duchenne paralysis).

Motor Symptoms.—The muscles affected by paralysis and wasting in this lesion are the *deltoid*, *biceps*, *brachialis anticus*, and *supinator longus*, as well as the *supraspinatus* and *infraspinatus*, *rhomboideus* and *subscapularis*, the clavicular fibres of the *pectoralis major*, *serratus magnus*, and, finally, the *latissimus dorsi* and *teres major*.

The most striking symptom, on the one hand, is that the arm cannot be flexed at the elbow on account of paralysis of the flexors of the forearm, nor can it be raised or abducted on account of paralysis of the deltoid; but, on the other hand, the movements of the wrist and fingers are not interfered with, because the functional activity of the muscles is quite unimpaired.

On closer examination it will be found that the arm is weak in adduction (pectoralis major), and that it can only be feebly rotated inwards or outwards (supraspinatus and infraspinatus), or not at all; and in an attempt to appose the two shoulder-blades, the bone on the affected side deviates

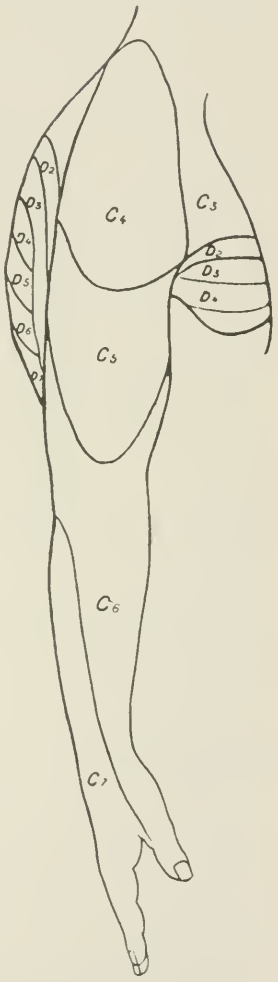


FIG. 55.—Sensory radicular distribution of upper limb (external aspect).

more from the middle line, and the rhomboid stands out less.

The patient can barely, or only feebly, put his affected hand on the buttock of the sound side. To test this weakness of the latissimus dorsi the muscle at the posterior border of the base of the axilla should be palpated and compared with the sound side.

It should be remembered that in these cases there is frequently ankylosis of the shoulder, which limits the movement of the joint.

Atrophy, which at first appears to involve the whole of the upper limb, is chiefly confined to considerable flattening of the shoulder, and the muscles of the supra- and infraspinous fossæ, biceps, and supinator longus.

Electrical Changes.—Electrical changes may be very marked at an early stage.

Reaction of degeneration is often complete in the deltoid, the anterior segment of the arm, and the supinator longus, and nearly so in the supra- and infraspinati. though it may be partial in the other muscles.

Sensory Symptoms.—Sensory disturbances affect a root distribution, and occupy a transverse band along the outer border of the arm, the forearm, and hand in the area of the fifth and sixth cervical roots (fig. 56).

They take the form of hypæsthesia to pricking, anæsthesia to slight touch, and often anæsthesia to cold, but more particularly to heat.

In the early stages this sensory disturbance seems to be so great that some patients complain that they cannot feel their arm at all, and even go so far "as to look for it in their bed," although there is no accompanying lesion of the cervical cord.

These sensory symptoms soon improve; but as the lesion disappears, "pins and needles" and burning sensations over the whole area affected by hypæsthesia, but principally in the thumb and index finger, are complained of.

Reflexes.—The reflexes are affected. The *stylo-*

radial reflex causes no contraction of the biceps, supinator longus, or deltoid muscles.

Percussion of the styloid process of the radius, in-

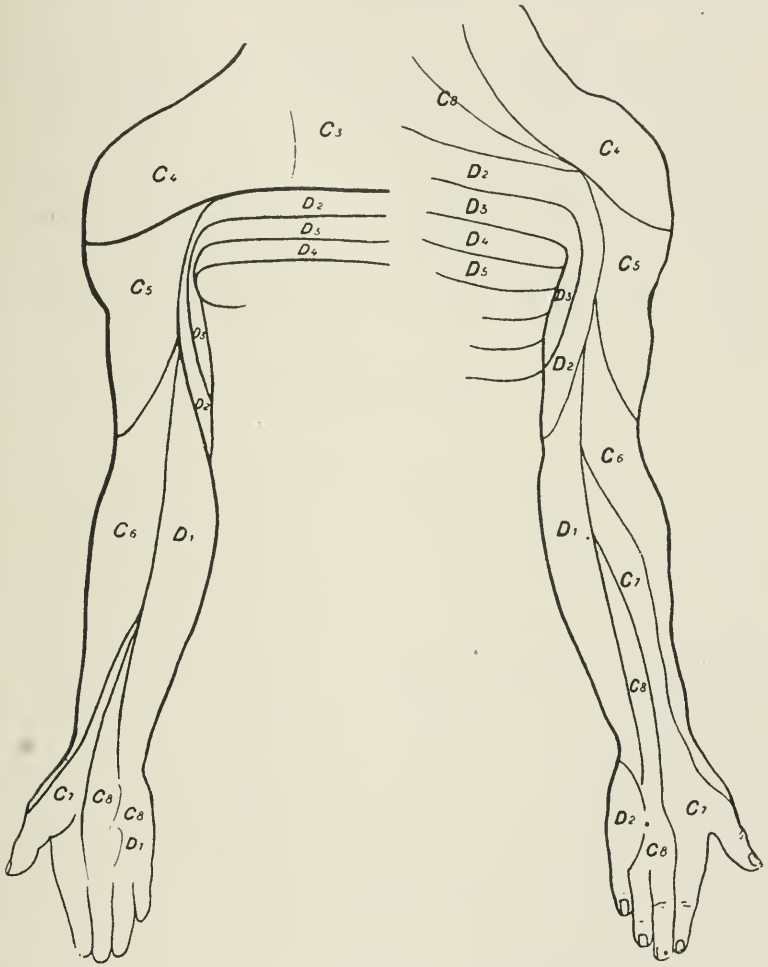


FIG. 56.—Sensory radicular distribution of the upper limb (ventral aspect on the left, dorsal aspect on the right).

stead of causing normal flexion of the forearm, produces a more or less energetic flexion of the fingers.

The *triceps reflex* is normal and sometimes brisk.

The *scapula reflex* produces no contraction of the deltoid or of the supra- and infraspinati, and frequently, too, contraction of the pectoralis major is absent.

Idiomuscular contraction, which is generally strong in all the muscles, becomes slow at last in the anterior segment of the arm, deltoid, and supra- and infraspinati.

Recovery.—Recovery of the power of movement begins in a few months, for in this upper root form, as in most of the lesions of the brachial plexus and its roots, there is a remarkable tendency to a complete or partial disappearance of motor and sensory disturbances.

The biceps and brachialis anticus seem to regain their functional activity quicker than the supinator longus.

To flex the forearm the patient raises his shoulder vigorously, stiffening all the muscles and leaning over to the sound side, and lifts his arm passively with the lateral wall of the thorax to supplement the defective movement of the deltoid. Then the forearm and hand creep down the side of the trunk, and, with maximum flexion, the hand will be as high as the sound shoulder, but cannot reach the shoulder on its own side. These first attempts at flexion of the elbow are imperfect and soon exhausted.

The rhomboideus, pectoralis major, latissimus dorsi, and teres major are not slow in recovering functional activity, for in them paralysis is sometimes only partial. The deltoid, supra- and infraspinati, and the supinator longus are the only muscles which take a long time to recover completely.

Faradic excitability and galvanic excitability return long before complete restoration of movement.

Sometimes involuntary movements of abduction and adduction of the thumb, of pronation and supination of the forearm, etc., are noticed whilst recovery of movement is taking place.

III. MIDDLE PLEXUS PARALYSIS

A third, and very common, variety of plexus lesion is paralysis of the musculo-spiral and circumflex nerve-roots (posterior cord), which causes total musculo-spiral paralysis, including the triceps, and complete paralysis of the deltoid muscles. A glance at Plate VI. will explain why paralysis of the latissimus dorsi so commonly occurs (as well as paralysis of the subscapularis and teres major), for the nerve supplying the latissimus dorsi arises from the musculo-spiral and circumflex trunk, or from the circumflex nerve direct.

Sometimes paralysis of distant muscles, such as the supra- and infraspinati, occurs simultaneously. The course of the projectile, the injury it does to bones and muscles, the pressure due to extravasation of blood, and the subsequent formation of cicatricial tissue account for this.

IV. COMPLEX PARALYSIS OF THE PLEXUS

In addition to the three simple and clearly-defined types of paralysis just mentioned there may be more complicated paralyses involving the whole, or nearly the whole, of the brachial plexus.

Careful examination of the functional activity of each segment of the upper extremity, and of each individual muscle, as well as investigation of the sensory disturbances in all the areas of the cervical and brachial plexuses, would be essential to gauge the extent and gravity of the lesion. Some months' observation, repeated clinical and electrical examinations, would enable the observer to estimate the recovery of a certain amount of function or the persistence in gravity of the lesion.

If there is no marked improvement (other circumstances being favourable), surgical intervention should be resorted to. Sometimes a nerve-trunk requires freeing from a callus of the clavicle or from cicatricial sclerosis, and at other times the divided nerves require suture.

We must, however, repeat that *lesions of the brachial plexus have a general tendency to recover*, and that recovery may be complete after the lapse of from six months to two years, according to the circumstances of the case. Often this recovery is only partial, and definite paralysis of one or two nerves, or of some muscles, may be permanent.

The following is a brief account of some cases, with their corresponding diagrams. They give some idea of the great variety of the lesions of the brachial plexus, and of the first four cervical roots or their collateral branches.

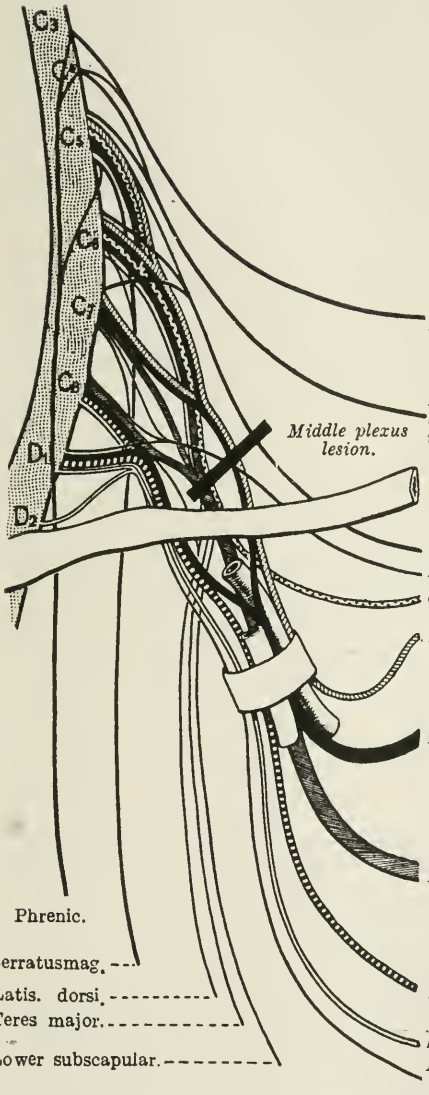
Case I.—Shrapnel bullet wound: point of entry, innermost part of the left infraspinous fossa; of exit, right subclavicular region 3 cm. from the lower border, and four fingers' breadths to the left of the sternal extremity of the clavicle. Wound of lung.

Immediate paralysis of left arm; total paralysis of the musculo-spiral and circumflex trunk, and of the musculo-cutaneous. Less marked paralysis of the supra- and infraspinati, rhomboideus, subscapularis, pectoralis major, and teres major. Paresis of the pronators and flexors of the wrist. Ulnar and internal cutaneous nerves, flexors of fingers and thenar muscles unaffected. Very marked hypæsthesia in the circumflex area, less marked in the musculo-spiral and in the forearm and hand.

Complete reaction of degeneration of the muscles supplied by the musculo-spiral and circumflex nerves and of the anterior segment of the arm. Partial reaction of degeneration of the pectoralis major, latissimus dorsi, supra- and infraspinati, teres major, pronator radii teres, and flexor carpi radialis; the flexor muscles of the fingers and thumb and the thenar muscles were more slightly affected.

Fig. 57 shows the probable localisation of the lesion in view of the course taken by the bullet and the clinical signs.

Case II.—Rifle bullet wound: point of entry just above the middle part of the right clavicle (fracture), three fingers' breadths to the right of the sternal head of the sterno-cleido-mastoid; point of exit, behind through the middle of the infraspinous fossa at three fingers' breadths above the inferior angle of the scapula. Immediate paralysis. Total paralysis of the musculo-spiral, circumflex, ulnar and internal cutaneous nerves. Partial paralysis of the flexor muscles of the fingers and thenar muscles. Paresis of the palmars and pronator radii teres, biceps, and brachialis anticus. Painful phenomena in the median area. Sensory disturbances in the musculo-spiral, ulnar and internal cutaneous regions.



LOCALISATION OF LESIONS IN BRACHIAL PLEXUS.

NERVES.	MUSCLES.	
Nerve to trapezius.	Trapezius.	
Nerves to scapular muscles.	Levator anguli scapulæ.	—
	Rhomboideus	—
	Supra- and infraspinatus	—
	Subclavius.	—
	Subscapularis sup.	—
	CLAVICLE.	
Ext. ant. thor.	Pectoralis major	—
Int. ant. thor.	Pectoralis minor	—
Circumflex.	Deltoid	O
Mus.-cutan.	Biceps	O
	Coraco-brachialis	O
	Brachialis anticus	O
Median.	Pronator radii teres	—
	Flexor carpi radialis	—
	Palmaris longus	—
	Flexor sublimis dig.	
	Flexor profundus (1 and 2).	
	Pronator quadratus.	
	Abductor brevis pollicis.	
	Flexor brevis pollicis.	
	Opponens pollicis.	
	I. and II. Lumbricales.	
Mus.-spiral.	Triceps. Anconeus.	O
	Supinator longus	O
	Ext. carpi rad. long. et brev.	O
	Extensor commun.	O
	Extensor prop. indicis	O
	Extensor minimi dig.	O
	Extensor long. et brev. poll.	O
	Adductor longus poll.	O
	Extensor carpi ulnaris	O
Ulnar.	Flexor carpi ulnaris.	
	Flexor profundus (3 and 4).	
	Hypothenars.	
	Interossei.	
	III. and IV. Lumbricales.	
	Adductor pollicis.	
Int. cutan.		
Lesser int. cutaneous.		

FIG. 57.—Henry Meige's diagram. The black line shows probable seat of lesion. Muscles totally paralysed are marked O, those partially paralysed —

Case III.—Injury by shrapnel bullet: point of entry, in the neck 3 cm. below the tip of the left mastoid process. The shell lodged at the back of the sternal notch. Penetrating wound of chest. The arm became stiff at once and then dropped down inert. Pain in the shoulder, in the arm, and on the outer side of the forearm, and also on the dorsal aspects of the thumb and index finger.

Paralysis of the trapezius, sterno-cleido-mastoid, supra- and infra-spinati, rhomboideus, levator anguli scapulæ, and deltoid. Paresis of the biceps, brachialis anticus, supinator longus, pectoralis major, and of the diaphragm (slight asymmetry of the two hypochondria in breathing). Objective sensory disturbances in the area of second, third, and fourth cervical roots; hyperæsthesia to pricking in the fifth and sixth cervical area. Complete reaction of degeneration of the supra- and infraspinati. Partial reaction of degeneration of the trapezius, sterno-mastoid, and deltoid. Slight faradic and galvanic hypoexcitability of the biceps, brachialis anticus, and supinator longus. (For seat of lesion, see fig. 58.)

Lesions of the plexus may be incomplete and variable, just as palsies of the peripheral nerves are frequently partial and dissociated. Paralysis which is total at the onset improves in a few weeks or months; some of the paralytic symptoms, particularly the sensory disturbances, disappear as the result of absorption of extravasated blood or the products of inflammation which in the early stages of injury press on the nerve-trunks.

The following is a brief description of a case in point:—

Case IV.—Rifle bullet wound: point of entry, below the left clavicle in the anterior wall of the axilla; point of exit, four fingers' breadths from the dorsal border of the scapula, about 5 cm. from its inferior angle. Penetrating wound of chest.

Immediate and total paralysis of arm; copious hæmorrhage from wound with secondary hæmorrhage.

On examination a year after injury: there is paralysis of the supinator longus, radial extensors, extensors of fingers and thumb, and abductor longus pollicis. Paralysis of the hypothenar muscles and interossei. Partial paralysis of the biceps, flexor carpi ulnaris, and thenar muscles. Still slighter paralysis of deltoid, triceps, palmars, flexor carpi ulnaris, and of the superficial and deep flexors. Much hypæsthesia to all kinds of sensation in the musculo-cutaneous region of the forearm and of ulnar in the hand. Slight disturbance of sensation of touch, heat and cold in the median, internal

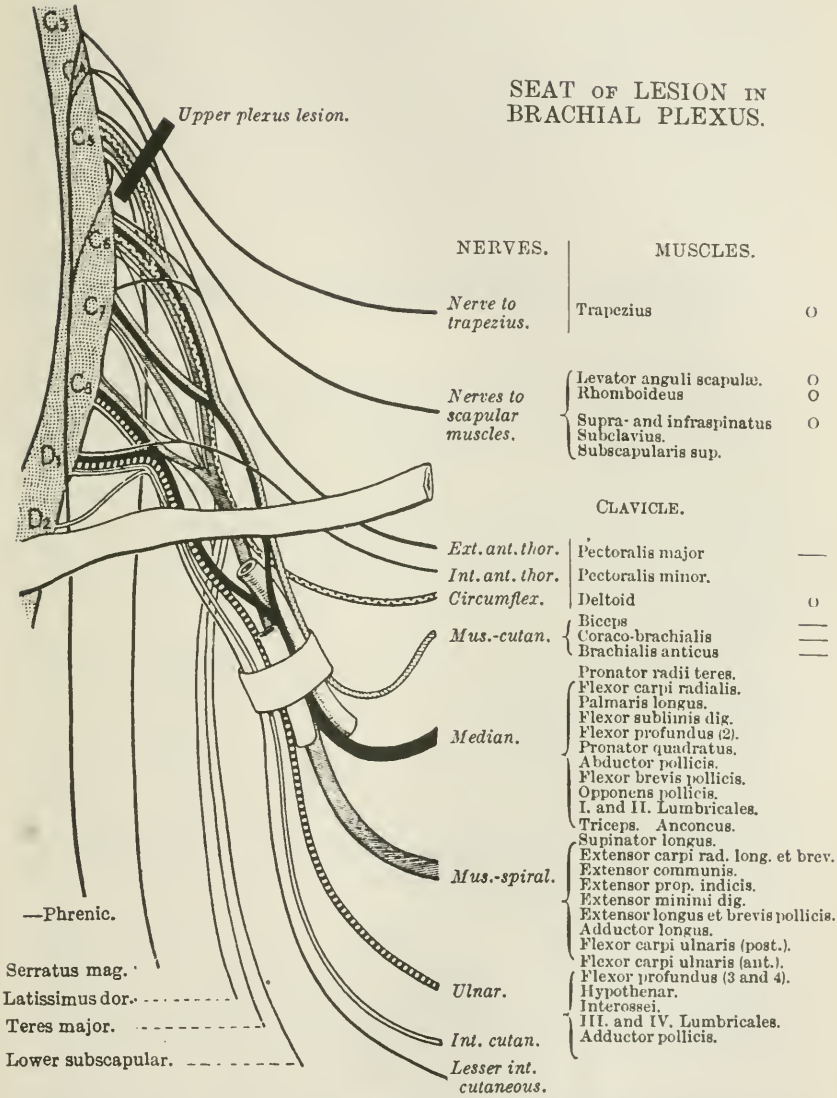


FIG. 58.—Henry Meige's diagram. The black line shows probable seat of lesion. Muscles totally paralysed are marked O, those partially paralysed —.

cutaneous, and musculo-spiral areas. The electrical changes were : faradic and galvanic hypoexcitability without marked slowness of the contraction. This lessened irritability was very marked in the deltoid, triceps, biceps, and supinator longus ; less marked in the palmars, flexor carpi ulnaris, extensor carpi ulnaris, the flexors of the fingers, thenar and interosseous muscles. Loss of faradic excitability and diminution of galvanic excitability in the extensors of fingers and thumb, radial extensors, abductor longus pollicis, hypothenar muscles, and last three interossei.

V. PARALYSIS OF THE BRACHIAL PLEXUS FOLLOWING DISLOCATION OF THE SHOULDER OR DRAGGING ON THE CERVICAL ROOTS

Some forms of total paralysis of the brachial plexus may be due to *dislocation of the shoulder* which has been reduced, or to dragging on the roots which form the plexus when the wounded man has been dragged along by the arm when it was raised and abducted. This form of paralysis comes on at once or supervenes a few hours after the accident, and is preceded by persistent " pins and needles " in the tips of the fingers.

Then suddenly the hand becomes numb and paralysed, and these sensory and motor changes extend nearly to the shoulder.

This type of case is known as *total paralysis* of the plexus, or rather of all its terminal branches including the circumflex, the other muscles of the shoulder supplied by the collateral branches being more or less unaffected : in all the cases observed by us they were not completely paralysed.

Sensory changes are confined to the hand, in which there is complete anæsthesia to all forms of sensation, with loss of bone sensibility and the sense of position ; in the forearm and in the lower part of the arm total anæsthesia goes higher up on the anterior than on the posterior aspect. The sensory disturbances become less intense towards the root of the limb. The elbow, hand, and fingers have lost all power of movement.

Movements are possible in the shoulder alone, but

they are weaker than normal and limited in extent; for there is always some stiffness in the shoulder, which may amount even to true ankylosis.

There were no signs of Claude Bernard and Horner's *sympathetic syndrome* in our cases.

The electrical changes consist in a reaction of degeneration, which is more marked and complete the nearer one gets to the hand, where faradic and galvanic loss of excitability is the rule.

This paralysis remains stationary some months before it begins to retrocede steadily from the shoulder downward.

The first nerves to recover are the circumflex and the musculo-cutaneous.

It takes eighteen months to two years for any perceptible recovery to take place. The nerves most affected are the ulnar and median. Paralysis for a very long time predominates in the small muscles of the hand, especially the interossei, the hypothenar, and some of the thenar muscles, so that the result at the end of eighteen months is paralysis of the ulnar nerve only.

As the affection develops, retraction of the tendons of the flexors begins, causing clawing of the last three or four fingers, which becomes more marked towards the little finger.

The sensory disturbances disappear in due course; paræsthesia appears with painful hyperæsthesia, particularly in the median area. Hypæsthesia in the cutaneous region of the ulnar nerve persists for a long time.

Spontaneous pain and pain from massage or movement of the limb may be fairly acute in the palm of the hand and along the course of the nerve-trunks.

Electrical recovery is rapid in muscles which have begun to regain their functions. The muscular atrophy gradually disappears.

Table I.—Showing Distribution of the Motor Nerves of the Cervical and Brachial Plexuses [1]

Roots.	Nerves.	Muscles.
C^1	Nerves to	Rectus capitis anticus major.
		Rectus capitis anticus minor.
		Rectus lateralis.
	Anastomosis with hypoglossal	Genio-hyoid and muscles of the sub-hyoid region.
C^2	Nerves to	Rectus capitis anticus major.
		Longus colli.
		Sterno-mastoid.
	Anastomosis with hypoglossal	Genio-hyoid and muscles of the sub-hyoid region.
C^3	Nerves to	Rectus capitis anticus major.
		Longus colli.
		Scalenus medius.
		Trapezius.
		Levator anguli scapulæ and rhomboideus.
		(Sterno-mastoid).
	Descendens hypoglossi	Muscles of the sub-hyoid region.
	Phrenic	Diaphragm.
C^4	Nerves to	Longus colli.
		Trapezius.
		Levator anguli scapulæ and rhomboideus.
		Scalenus medius.
		(Scalenus anticus.)
	Phrenic	Diaphragm.
C^5	Nerves to	Longus colli.
		Scaleni.
		Levator anguli and rhomboid.
		Serratus magnus.
		Subclavius.
		Subscapularis.
	Suprascapular	Supra- and infraspinatus.
	Circumflex	Deltoid, teres minor.
	Musculo-cutaneous (Accessorily.)	Biceps, brachialis anticus.
	Musculo-spiral	Triceps (?), extensors.
	Phrenic	Diaphragm.
	Nerves to	Pectoralis major.
		Teres major.

[1] Poirier et Charpy, tome iii. fascicule 3.

Roots.	Nerves.	Muscles.
C⁶	Nerves to	Longus colli.
		Scaleni.
		Subscapularis.
		Teres major.
		Pectoralis major.
	Circumflex	Deltoid.
	Musculo-cutaneous	Biceps, brachialis anticus
	Median	Pronator radii teres.
		Flexor carpi radialis.
		Thenar muscles.
	Musculo-spiral	Triceps.
		Supinator longus et brevis.
C⁷	Nerves to	Scalenus medius.
		Latissimus dorsi.
		Subscapularis.
	Thoracic nerves	Pectoralis major et minor.
	Musculo-cutaneous	Coraco-brachialis.
	Median	Flexor sublimis digitorum.
		Flexor profundus digitorum.
		Flexor longus pollicis.
		Pronator quadratus.
		Thenar muscles.
	Musculo-spiral	Triceps.
		Anconeus.
		Extensor carpi ulnaris.
		Extensor communis digitorum.
	Ulnar	Flexor carpi ulnaris.
		(Flexor profundus.)
		III. and IV. Lumbricales.
	Nerves to	Serratus magnus.
		Teres major.
C⁸	Nerves to	Longus colli.
		Latissimus dorsi.
	Thoracic nerves	Pectoralis major.
		Pectoralis minor.
	Median	Flexor sublimis digitorum.
		Flexor profundus digitorum.
		I. and II. Lumbricales.
	Musculo-spiral	Triceps.
		Anconeus.
	Ulnar	Flexor carpi ulnaris.
		Flexor profundus III. and IV.
		Hypothenar muscles.
		Adductor pollicis.
		Interossei.

Roots.	Nerves.	Muscles.
D^1	Thoracic nerves	Pectoralis minor.
	Median	Flexor sublimis digitorum. Flexor profundus digitorum. Pronator quadratus.
	Ulnar	Flexor carpi ulnaris. III. and IV. Lumbricales.
	First intercostal	Intercostals. Levatores costarum. Serratus posticus superior.

Spinal Cord Symptoms associated with Lesions of the Brachial Plexus

We have already stated that lesions of the brachial plexus are often accompanied at their onset by paralysis of the whole arm, in which the motor and sensory symptoms soon subside; but grave lesions, or cases in which the nerve has been divided, show no signs of recovery.

We desire now to direct attention to plexus lesions (cervical roots, primary and secondary trunks) complicated by lesions of the spinal cord, which are sometimes difficult to detect.[1] These plexus or root lesions accompanied by spinal injury may run their course in two different ways:—

A. EARLY AND GENERALISED FORM

In this type there is almost always injury by a projectile which has passed right through the neck, either from left to right or from right to left. The symptoms which appear at once are, that the patient falls forward without losing consciousness, and *all the four limbs are paralysed.* Sometimes the sphincters of the bladder and rectum are paralysed, causing incontinence of urine and fæces.

A few hours or a few days after injury *pain* comes on in all the four limbs, principally at the roots of

[1] M. Pierre Marie and Mme. Ath.-Benisty, "Remarques cliniques sur quelques cas de lésions de la moelle cervicale par plaies de guerre," *Soc. Médicale des Hôpitaux*, June 11, 1915, et *Soc. de Neurologie*, December 2, 1915.

the nerve, more especially in the arm. This pain is sometimes unbearable, and the prolonged use of opiates becomes necessary.

Paralysis soon develops. A few days or weeks

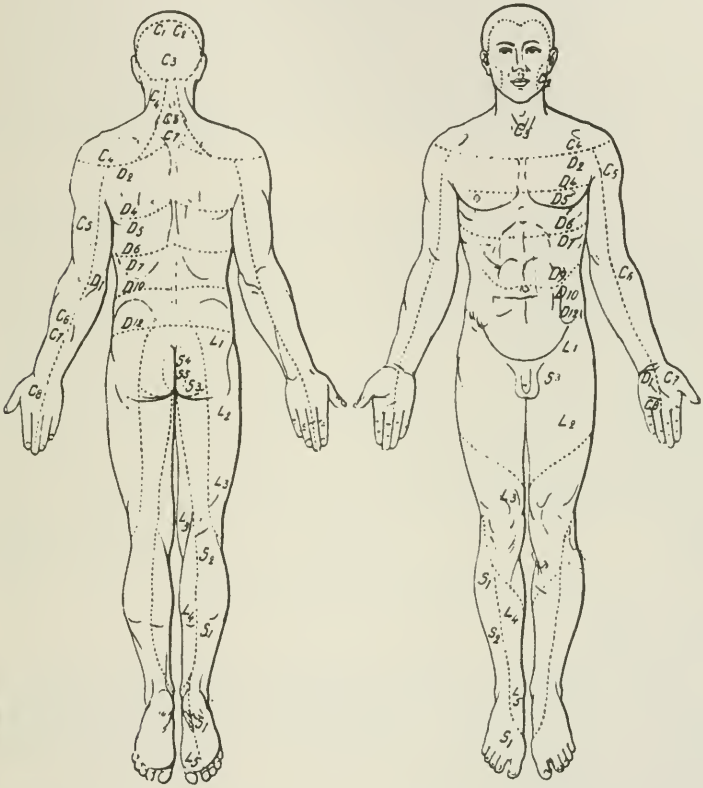


Fig. 59.—Dorsal aspect. Fig. 60.—Ventral aspect.

Figs. 59 and 60.—Root innervation of trunk and extremities.

after the accident, according to the gravity of the injury, the lesion subsides in one entire half of the body. From quadriplegia it develops into hemiplegia; then the leg on the affected side recovers its movements, until *brachial monoplegia* alone remains, which is often very severe, and persists for a long time, in serious cases improving only slowly,

but disappearing in a few months in slight cases. Pain follows the course of the motor symptoms, and some months after the injury it is only complained of in the affected arm.

The *sphincter troubles* vary; they may improve gradually from the first, or persist for weeks or months, and in that event deep bedsores may form.

Though the patient's condition steadily improves, some months or even a year after injury very definite signs of affection of the spinal cord are still present.

In the early stages these consist of an obvious exaggeration of the tendon reflexes, which are more brisk on one side, together with unilateral or bilateral *Babinski's sign*, ankle clonus, disturbances in co-ordinated action, synkinesis, and especially *Brown-Séquard's syndrome*, which, though not very marked, is always present, and is frequently accompanied by a definite syringomyelic dissociation on the side which is least paralysed.

Sensibility to cold is generally most affected. Sensibility to heat is accompanied by painful hyperæsthesia; the sense of position and bone sensibility are sometimes definitely diminished on the paralysed side.

As regards brachial monoplegia, the loss of power is most marked in the hand and fingers, though it affects the different muscles to a varying degree, and is accompanied by electrical changes greater in quantity than quality.

In some cases the seat of the lesion is the pyramidal tract, as shown by contracture and exaggeration of reflexes; sometimes it is in the nerve-roots, as evidenced by flaccidity, loss of reflexes, and a tendency to reaction of degeneration. But most commonly it is a combined lesion of the pyramidal tract, marked by exaggeration of reflexes, combined with affection of the nerve-roots, shown by bands of anæsthesia or hypæsthesia, flaccid paralysis in some muscles supplied by the same root, spontaneous pain in the zones of

one or several roots, and pain on pressure on the nerve-trunks.

A final detail should be added to this clinical picture, viz. the frequent presence of *Claude Bernard and Horner's sympathetic syndrome* (enophthalmos, narrowing of the palpebral fissure, and myosis), most commonly on the paralysed side, and very frequently intermittent and disappearing entirely on some days without any particular reason. Perspiration is sometimes diminished on the face on the side where the ocular phenomena occur.

This *sympathetic syndrome* is due either to an actual lesion of the cervical cord of the sympathetic, or to pressure, or to a lesion of some kind, of the upper dorsal cord or of the first dorsal nerve-root.[1]

Skiagrams do not usually show any disease of bone.

Considering the rapid retrocession of the spinal symptoms, this early type would appear to be due to a kind of commotion of the spinal cord, combined with more or less severe lesions of some of the cervical nerve-roots.

B. PARTIAL AND PROGRESSIVE FORM

In this type of case the clinical appearances are not so simple ; the spinal affection is not so easy to demonstrate, and its pathogeny is obscure.

The wound is *lateral*, the projectile having passed from front to back or from back to front, to the left or the right of the median axis of the neck. Consciousness is almost always lost at once, and not regained for some considerable time ; this condition is accompanied sometimes by temporary aphonia and deafness, but without any sphincter trouble. As soon as the patient recovers consciousness he finds that his limbs are not paralysed at all, with the exception

[1] Faure-Beaulieu has related a case of Brown-Séquard's syndrome due to a wound of the cervical cord, with an alternating inequality of the pupils, or " see-saw myosis." *Soc. de Neur.*, May 4, 1916. *Rev. Neurol.*, June 1916, p. 948.

of the arm on the side of the wound, which is always absolutely powerless and insensitive. The clinical picture is that of *a complete lesion of the brachial plexus*, with total paralysis, anæsthesia reaching to the shoulder, and loss of reflexes.

But this clinical appearance soon changes, for pain appears, which at the onset is generalised in the whole arm, or localised from the first to the course of some of the roots. Simultaneously the paralysis begins to retrocede, and sometimes so rapidly that movements are recovered in a few weeks, reflexes reappear and then become exaggerated; paralysis, which was slight at first, develops into a deformity, which becomes worse, until the limb has all the appearance of the common type of hemiplegic arm.

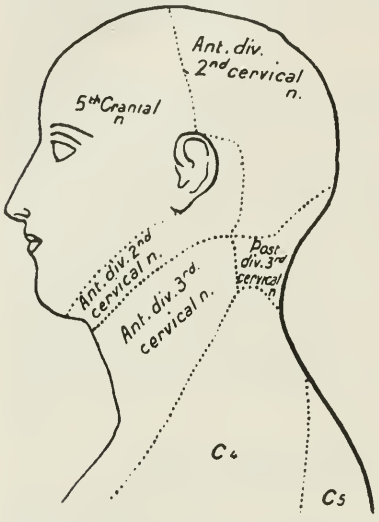


FIG. 61.—Sensory radicular distribution of head and neck.

But more often recovery of movement is slow, the paralysis remaining flaccid for a long time.

The first movements which return are those of the fingers (slight flexion), and then slight movements appear in the wrist, elbow, and shoulder.

Anæsthesia, which was total at first, is confined to the zone of one or more nerve-roots. The electrical reactions are definitely affected, reaction of degeneration being found in some of the muscles supplied by one or two anterior roots.

But spastic symptoms appear simultaneously. The triceps reflex is lost, and there is paralysis of the triceps and palmar muscles (C^7), whilst the stylo-radial wrist reflex is brisk. Pain generally referred to the area of

impaired sensation, or to the zone of the nerve-roots immediately above or below. Pressure on the nerve-trunks is painful.

Paralysis of the upper extremity goes on developing in this way, until one day the patient complains of the leg on the affected side.

He feels a weakness in the lower limb ; his foot drops, and sweeps the ground as he walks ; he develops a steppage gait, or circumducts his leg like the subject of hemiplegia ; or again the movements of his leg

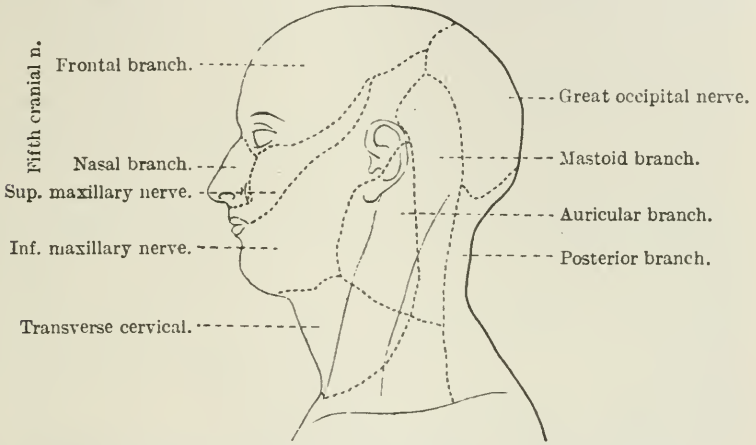


FIG. 62.—Sensory peripheral distribution of head and neck.

are inco-ordinate, like a person with ataxia. Examination brings out definite *but very variable* symptoms of *spinal injury* ; sometimes Babinski's sign is present ; at others, there is ankle or patellar clonus.

The tendon reflexes are, however, always brisker than those on the opposite side ; the cremasteric and the abdominal are, on the other hand, weaker or lost. The strength of the muscles is sometimes unaffected ; at others, paralysis attacks a group of muscles (extensors of the foot or the peronei). Sensory disorders, when present, are hyperæsthesia to pricking on the thigh, and sometimes diminution of bone sensibility and sense of position. Pressure on the muscles of the leg is often painful.

On the opposite side to the lesion more definite sensory changes are found, corresponding to Brown-Séquard syndrome, very commonly accompanied by a syringomyelic dissociation—*i.e.* hypæsthesia to pricking, anæsthesia to cold, and hypæsthesia, or, on the contrary, painful hyperæsthesia to heat.

The sensory disturbance generally extends to the nipple line, and in the arm occupies root areas—most commonly the area of the eighth cervical and first dorsal. At the limit of the zone of hypæsthesia to pricking there is frequently a band of hyperæsthesia in which pain is felt spontaneously, or on pricking.

As this paretic or spastic condition develops and becomes established in the leg on the injured side, the patient will suddenly one day complain of weakness of his arm on the opposite side to the injury. This weakness is usually limited to movements of clasping, or pressure of the hand and flexion of the elbow; at other times it is most marked in the same muscles as are most severely attacked in the wounded arm. Thus in a case in which the triceps and the flexor carpi radialis were the most affected in the paralysed arm, the patient's sound arm showed weakness in extension of the elbow and flexion of the hand.

Simultaneously with this paresis the reflexes are exaggerated, and there is slight inco-ordination, but—what is more significant—there is *loss of the stereognostic sense*, which is either complete, or less commonly limited to the outer or inner side of the hand.

What becomes of these symptoms which have been superadded to the initial brachial monoplegia, and where does this progressive involvement of the cord stop?

On the sound side the symptoms of paresis diminish and then disappear, as also do some of the spasmodic symptoms (ankle and patellar clonus), and Babinski's sign, but the tendon reflexes still remain brisk. In the sound hand weakness diminishes, the stereognostic sense becomes normal again, and the pain and "pins and needles" disappear.

On the affected side paralysis of the arm gradually improves, but symptoms of spinal and nerve-root mischief still persist.

The most common phenomena during this retrogressive stage are *muscular contractures* localised in a single muscle, or, less frequently, affecting a whole group ; there may be *contracture of the trapezius* on the wounded side, with hardening of the muscle and increased contraction on mechanical percussion and to electric currents ; *contracture of the pectoralis major* on the sound side or on the injured side, with the same physical signs ; and *contracture of the scaleni*. With these various phenomena there is aching in the contractured muscle, which causes great discomfort and inconvenience to the patient.

Inclination of the head towards the affected side, or, less commonly, towards the sound side, with displacement and rotation of the neck, is probably attributable to muscular contractures of this kind in the muscles of the back of the neck, trapezius, scaleni, and muscles of the upper segment. This inclination of the head remains permanent.

Spasmodic and involuntary movements may appear in the fingers or in the elbow of the affected side—such as flexion of the fingers, abduction, opposition of the thumb, sudden flexion of the forearm on the arm, pronation, and supination. Sometimes fibrillary contractions are seen in some of the affected muscles.

In this second type of spinal lesion the syndrome of Claude Bernard and Horner is less common.

Disturbances in perspiration are common ; there may be copious sweating on the sound hand, or in the axilla on the affected side, while the skin of the affected hand will be very dry.

Symptoms of paresis in the lower extremity on the wounded side and in the arm on the opposite side may reappear. We have come across a case in which, even after the lapse of six months, paresis reappeared in the left leg (the affected side), with greatly exaggerated tendon reflexes, ankle clonus, and paralysis of the

peronei ; whilst in the first attack there had been paralysis of the extensors of the foot and toes, with patellar clonus. A *lumbar puncture* performed at this time showed hypertension of the cerebro-spinal fluid, with slight increase in albumen, but without any cellular change.

Sensory symptoms on the opposite side to the injury disappear slowly, and the total hypæsthesia noted at first is followed by painful hyperæsthesia to cold or to heat, as the case may be, with diminished or altered perception of temperature.

In these two types of affection of the spinal cord associated with lesions of the nerve-roots of the brachial plexus there is some *rigidity of the neck*, with nuchal pain, especially when the patient changes his position in lying down or getting up. As skiagrams do not generally show any disease of bone, this rigidity may be attributable to lesion of the ligaments, and particularly to painful contracture of some of the muscles of the neck.

Most of the patients also complain that when they bend their neck they feel creeping sensations which are sometimes painful all over both arms, or localised in the epigastrium, or with a girdle distribution.

CHAPTER IX

GREAT SCIATIC, INTERNAL AND EXTERNAL POPLITEAL NERVES

THOUGH lesions of the anterior crural nerve and of its terminal branches are very rare, injuries of the great sciatic and its external branch, the external popliteal, are nearly as common as lesions of the musculo-spiral nerve.

It is very seldom that a wound of any depth of the posterior segment of the thigh leaves this nerve unaffected.

We shall first describe lesions involving the nerve-trunk of the great sciatic from where it emerges, in the true pelvis, to its bifurcation in the popliteal space.

But before doing this it will be necessary to give a short description of the anatomy of this nerve and of its terminal branches.

ANATOMY

The *great sciatic nerve* passes out of the pelvis and appears in the middle of the gluteal region, almost on the level of a horizontal line passing along the upper border of the great trochanter. At its origin it is covered by the lower half of the gluteus maximus, whence it descends to the thigh through the muscles on the posterior aspect of the limb, and covered by the long head of the biceps. It passes between the long head of the biceps externally, the semimembranosus and the semitendinosus internally, and comes out into the popliteal space. In its passage through the thigh the great sciatic nerve has a nutrient artery on one side of it—the *artery of the great sciatic*, which is a branch of the sciatic artery.

At the level of the gluteal fold the nerve gives branches to the posterior muscles of the thigh—viz. the *biceps, semimembranosus, semitendinosus,* and *adductor magnus.*

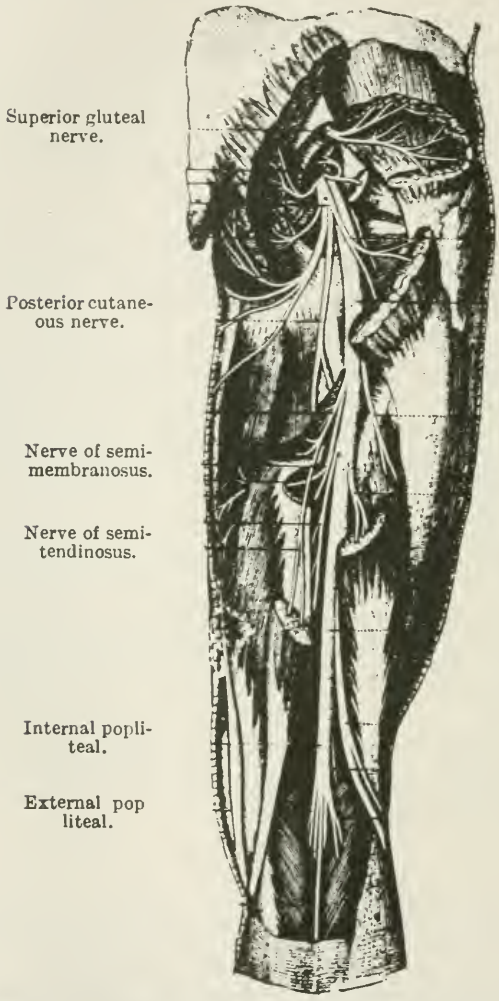


FIG. 63.—Sciatic nerve and its collateral branches in the buttock and thigh. (After Sappey.)

On reaching the popliteal space it divides into two terminal branches, the external and internal popliteal. Sometimes, however, the nerve divides much higher up in the gluteal region. As a

rule, a glance at the nerve will show it to be formed of two trunks in close juxtaposition, but separated by a longitudinal groove.

The *external popliteal nerve*, the outer branch of the bifurcation of

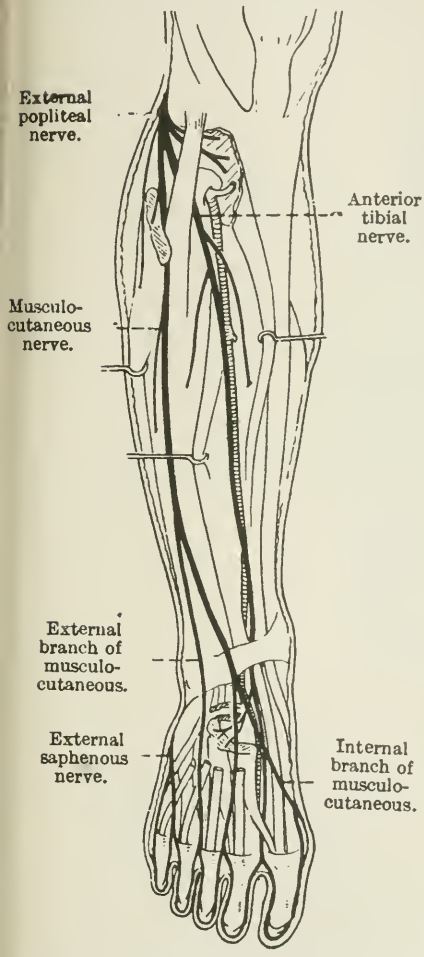


FIG. 64 —Musculo-cutaneous and anterior tibial nerves.

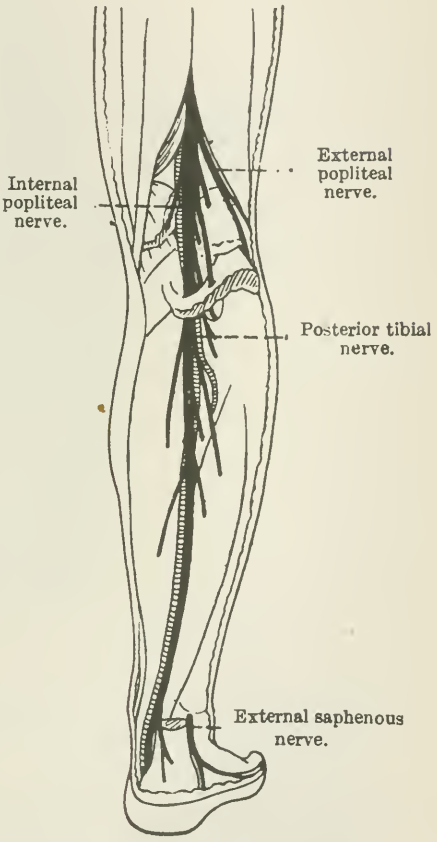


FIG. 65.—Internal popliteal and posterior tibial nerves.

the great sciatic, arises from the upper angle of the popliteal space. It passes along the tendon of the biceps and behind the head of the fibula, winding round the neck of this bone, which is the spot chosen for palpation of this nerve, and then ends by dividing within the upper insertions of the peroneus longus (fig. 64).

Its collateral branches are an articular branch for the knee, the accessory nerve of the saphenous or *communicans peronei*, the external *cutaneous peroneal nerve*, and some very slender muscular branches for all the upper part of the *tibialis anticus*; these last branches are given off very low down at the neck of the fibula, and

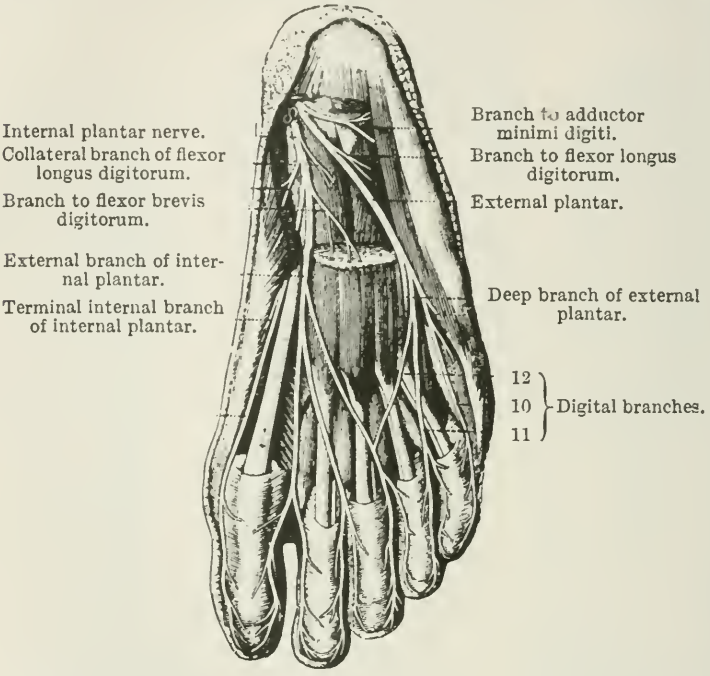


FIG. 66.—Plantar nerves, superficial region. (After Sappey.)

pass beneath the extensor longus digitorum to reach the muscle which they are intended to supply.

The terminal branches of the external popliteal are the musculo-cutaneous and anterior tibial.

The *musculo-cutaneous nerve* passes along the outer side of the fibula. At first it lies in the substance of the peroneus longus, then in the space which separates this muscle from the peroneus brevis; then it passes through the aponeurosis, becoming subcutaneous at three or four fingers' breadths above the tip of the external malleolus, and supplies the skin of the dorsal aspect of the foot.

In its course the musculo-cutaneous nerve supplies muscular branches to the *peroneus longus* and the *peroneus brevis*.

The *anterior tibial nerve* is the inner branch of the bifurcation of the external popliteal. Like the musculo-cutaneous nerve it rises

at the neck of the fibula, then turning inwards passes through the upper insertions of the peroneus longus and extensor longus and reaches the outer side of the anterior tibial artery; it then runs vertically and deeply on the interosseous membrane between the

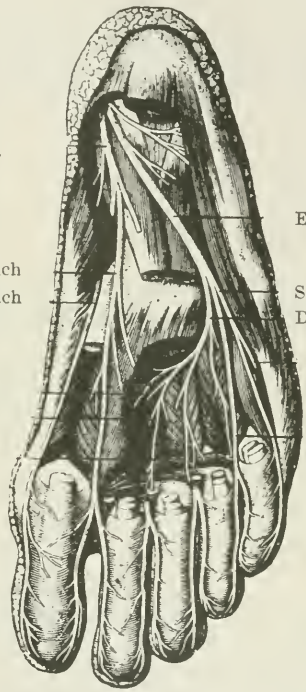


FIG. 67.—Plantar nerves, deep region. (After Sappey.)

tibialis anticus internally, and the extensor longus digitorum and then the extensor proprius hallucis externally.

Still accompanied by the anterior tibial artery at the ankle it passes under the anterior ligament of the tarsus, and terminates by bifurcating immediately below it.

In its course it supplies muscular branches to the *tibialis anticus*, *extensor longus*, *extensor proprius*, and *peroneus tertius*.

It ends in two branches, an *external*, which passes between the bones of the tarsus and the *extensor brevis digitorum*, which it supplies, and an *internal*, which descends vertically into the first dorsal interosseous space and supplies its cutaneous integument.

The *internal popliteal nerve*, the inner and larger of the two terminal branches of the great sciatic, descends through the middle of the popliteal space, then beneath the gastrocnemius and the arch

of the soleus to become the posterior tibial. In its course it runs close to the popliteal vessels and becomes relatively superficial, being covered only by aponeurosis, subcutaneous cellular tissue, and the outer skin (fig. 65).

Its muscular branches supply the *two heads of the gastrocnemius*, the *popliteus*, *plantaris*, *and soleus*; its articular branches supply the knee, and finally, an important sensory nerve—the *external saphenous* or *communicans tibialis*, which unites with the communicans peronei.

The *external saphenous* arises from the middle of the popliteal space and passes downward between the heads of the gastrocnemius to the outer margin of the tendo Achillis. At the lower third of the leg it supplies branches to the skin of the outer part of the leg, and ends on the outer side of the back of the foot.

The terminal branch of the internal popliteal—the *posterior tibial nerve*—commences at the arch of the soleus, and ends at the posterior aspect of the internal malleolus, where it divides into the *external* and *internal plantar nerves.*

In its upper two-thirds it runs very deeply in the posterior aspect of the leg, covered by the gastrocnemius and soleus in company with the posterior tibial artery. In its lower third it becomes more superficial, and is parallel with the inner margin of the tendo Achillis.

It supplies the *popliteus*, *tibialis posticus*, *flexor longus digitorum*, *flexor longus hallucis*, and a nerve to the deep surface of the *soleus.* Its sensory branches are the *calcaneo-plantar* and the *internal plantar nerve.*

The *internal plantar* (the homologue of the median nerve in the hand) supplies muscular branches to the *adductor hallucis*, *flexor brevis hallucis*, *flexor brevis digitorum*, *flexor accessorius*, and *first two lumbricales.*

Its sensory branches are the first seven *collateral plantar* branches.

The *external plantar nerve*, the smaller of the two (the homologue of the ulnar nerve in the hand), supplies all the *interossei*, *the muscles of the little toe*, *the third and fourth lumbricales*, *the abductor obliquus and transversus*; it also supplies the *last three collateral plantar branches.* As in the case of the ulnar in the hand, it has a *cutaneous branch*, which is purely sensory, and a *deep* muscular branch.

PHYSIOLOGY

The Muscles supplied by the Great Sciatic.—The *biceps*, *semitendinosus*, and *semimembranosus* are all flexors of the leg on the thigh.

According to Duchenne (of Boulogne), these muscles also extend the thigh on the pelvis in walking, and rotate the leg on the thigh, the semitendinosus rotating it inwards and the biceps outwards.

Muscles supplied by the External Popliteal.—The *tibialis anticus* causes very powerful flexion of the dorsum of the foot. Its thick

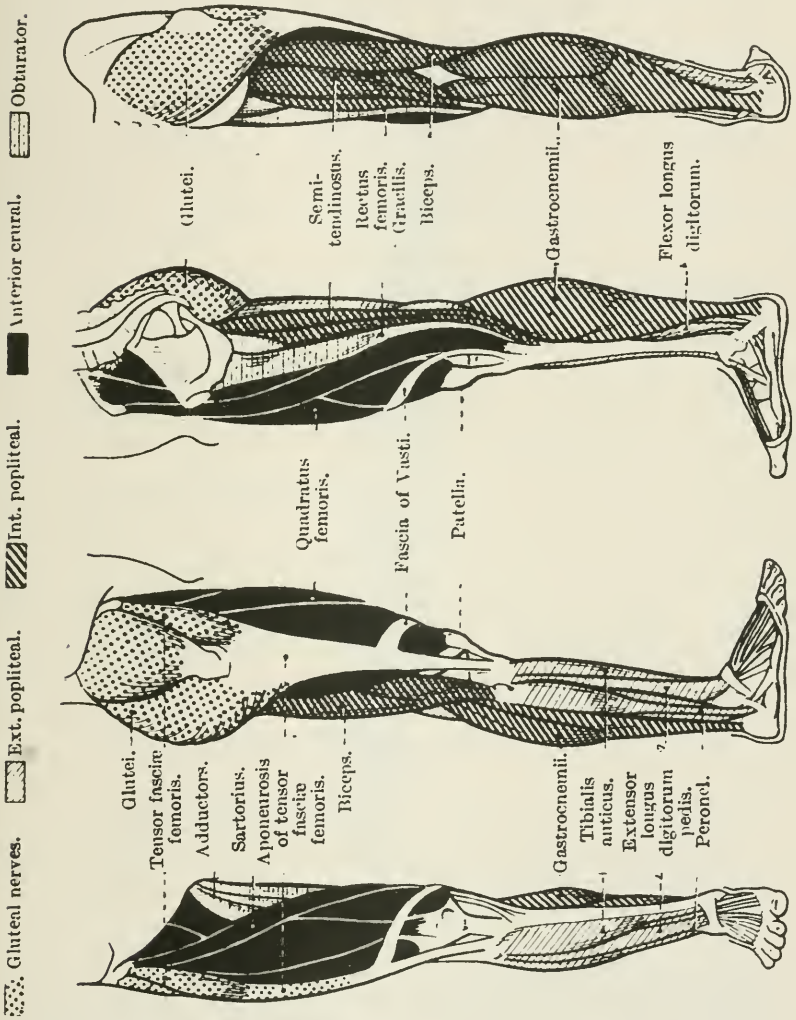


FIG. 68.—Nerve supply to muscles of the lower extremity.

tendon stands out prominently on the anterior aspect of the ankle when the foot is lifted and carried inwards, so that this muscle also adducts and rotates the foot.

The *extensor longus digitorum* extends the first phalanges of the

four outer toes. It assists in dorsal flexion of the foot, which it also abducts slightly.

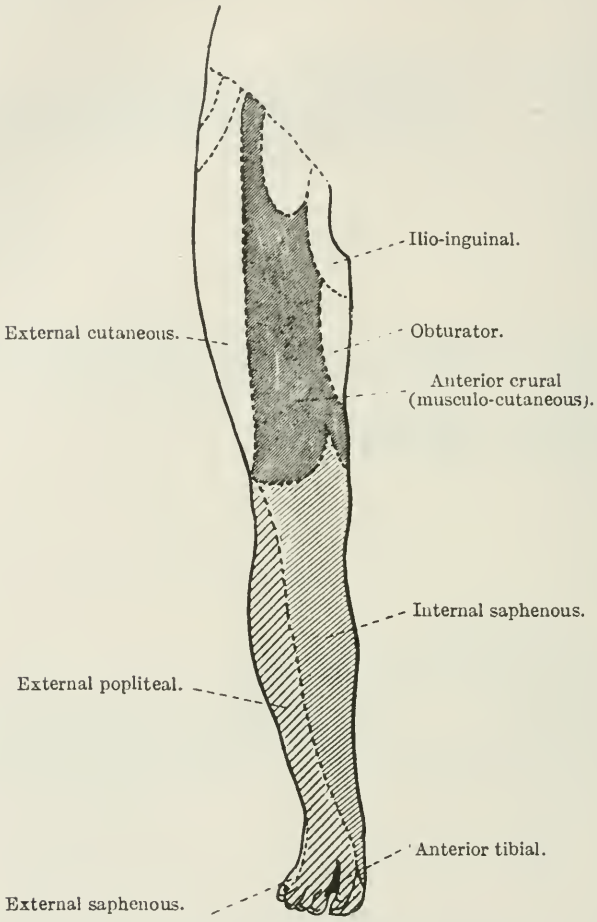


Fig. 69.—Peripheral sensory distribution of the lower extremity (anterior aspect).

The *extensor proprius hallucis* forcibly extends the first phalanx of the great toe, and the second very slightly.

The *peroneus tertius* is an inconstant muscle, and is only a part of the extensor longus.

The *peroneus longus* has the following action :—

1. It produces plantar flexion of the foot, but not so effectively as the gastrocnemius and soleus.

2. It lowers the inner side of the ball of the foot, making the plantar arch.

3. It gives the foot a movement of *abduction* and *rotation*, turning the foot outwards as its outer border rises.

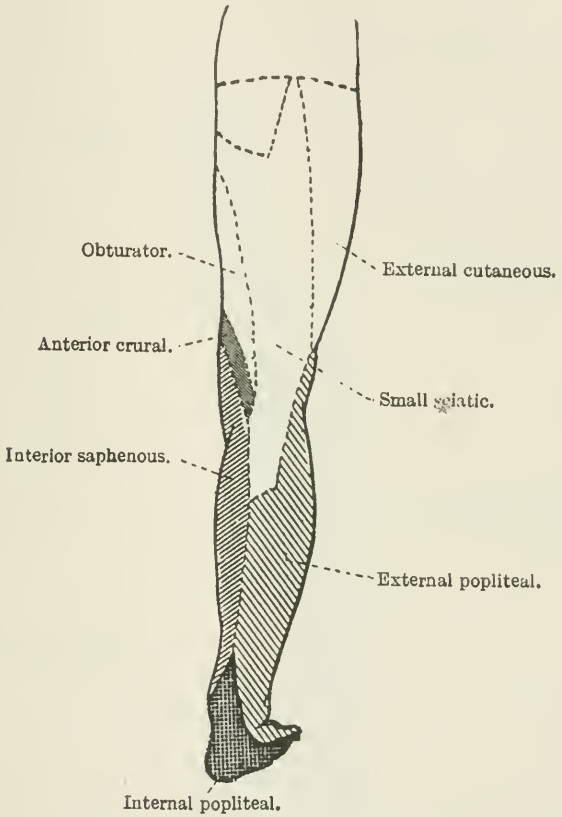


FIG. 70.—Peripheral sensory distribution of the lower extremity (posterior aspect).

The *peroneus brevis* abducts and everts the foot.

Finally, the *extensor brevis digitorum* assists the extensor longus, and extends the first phalanges of the first four toes.

Muscles supplied by the Internal Popliteal Nerve.—The *popliteus* muscle flexes the leg on the thigh.

The *plantaris* is very inconstant, and has no special action.

The *gastrocnemius* and the *soleus* unite to form a single muscle, the triceps of the leg, which ends in the powerful tendo Achillis. It

lowers the foot and enables it to prolong the leg, or rather to flex the sole of the foot in adduction, for it acts forcibly on the heel and on the outer half of the foot.

The peroneus longus being a flexor and abductor neutralises the

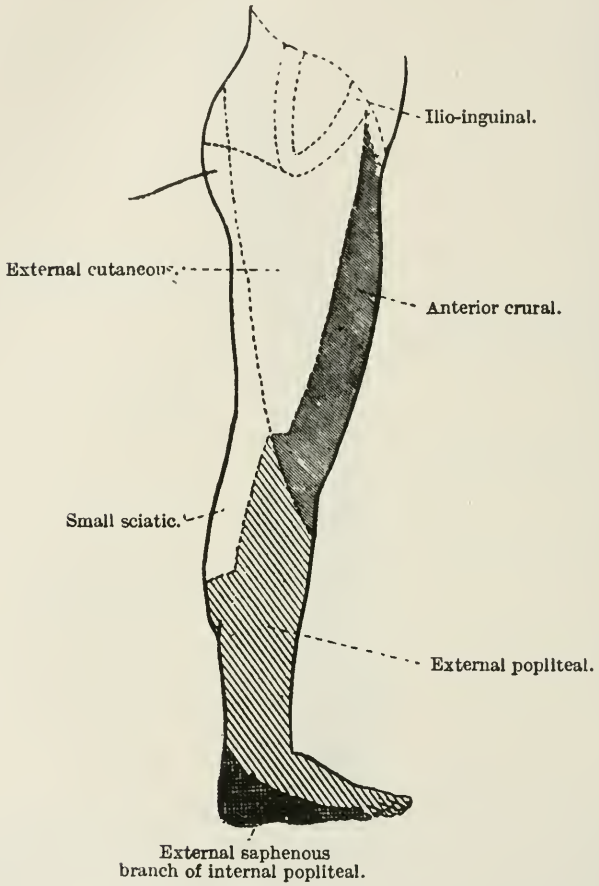


Fig. 71.—Peripheral sensory distribution of the lower limb (external aspect).

effect of the action of the triceps. Both contribute to direct plantar flexion of the foot.

Muscles supplied by the Posterior Tibial Nerve.—The *tibialis posticus* turns the foot directly inwards, being purely an adductor. As it contracts, its tendon can be seen standing out just behind the internal malleolus.

The *flexor longus digitorum* flexes the last two phalanges of the four outer toes.

The *flexor longus hallucis* flexes the second phalanx of the big toe.

Muscles supplied by the Plantar Nerves.—We shall divide the

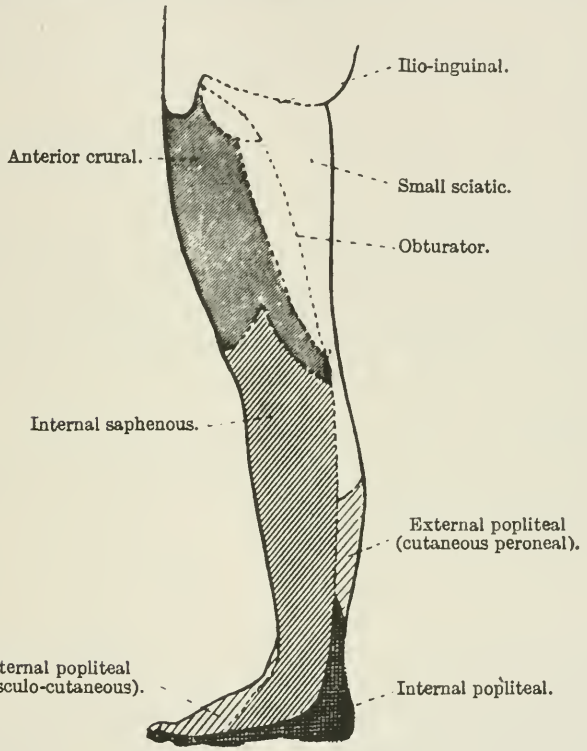


FIG. 72.—Peripheral sensory distribution of the lower limb (internal aspect).

muscles of the plantar region into three groups, those of the internal, external, and middle plantar regions.

The *internal region* corresponds to the thenar eminence in the hand. From the superficial to the deep layers there are three muscles :—

The *adductor obliquus hallucis*, which separates the big toe from the second.

The *flexor brevis hallucis*, which flexes the first phalanx on the corresponding metatarsal bone.

The *abductor hallucis*, which is inserted at the outer side of the base of the first phalanx of the big toe.

The abductor hallucis undoubtedly corresponds to the adductor pollicis in the hand. It flexes the big toe on the metatarsus and turns it outwards, bringing it near to the second toe.

The *external plantar region*, which corresponds to the hypothenar eminence in the hand, contains three muscles—*abductor minimi digiti*, *flexor brevis minimi digiti*, and *opponens minimi digiti*; all three flex the little toe on its metatarsal bone.

The *middle plantar region* from the superficial to the deep layers contains:—

The *flexor brevis digitorum*, which corresponds to the flexor sublimis digitorum in the hand; it flexes the second phalanx of the outer four toes.

The *flexor accessorius* assists the flexor longus digitorum.

The *lumbricales* are similar to the lumbricales in the hand.

The *interossei* have three plantar and four dorsal branches.

The lumbricales and the interossei, as in the hand, are the flexors of the first phalanx of the toes and the extensors of the last two phalanges. The interossei also undertake lateral movements of the toes, the plantar adduct the outer three toes, and the dorsal are abductors.

Briefly, the muscles in the sole of the foot (excepting the long and short flexors of the toes) simultaneously produce flexion of the first with extension of the second and terminal phalanges, without which action walking would be impossible.

Clinical Forms

The lesions of the great sciatic nerve are total or partial.

Partial lesions often assume painful forms, which we shall study in a special chapter, as we did with regard to the painful forms of the median nerve injuries.

A. TOTAL PARALYSIS

When the projectile has completely divided or caused a grave lesion of the nerve above the bifurcation, there follows total paralysis of all the muscles of the leg and foot.

The wounded fall prostrate the moment they are hit. Some have acute pain in the foot; others feel as if the limb were being torn away; others believe the leg is gone, or a sensation of numbness and cold pervades the whole leg from the periphery to the hip.

When they were able to get up and walk, supported by comrades, some noticed that the foot was dropped and inverted.

We have not had the opportunity of observing patients wounded in the sciatic nerve until six or eight weeks after their injuries. By this time suppuration had generally ceased, and they had already tried to walk with crutches or sticks, some even without any support.

Appearance of the Lower Limb.—The foot dangles and drops. In walking the knee is unduly raised, but the high steppage and the inclination to dropped foot are less accentuated than is the case when the external popliteal nerve alone is the seat of injury.

The anterior tibial and extensor tendons, so noticeable on the sound side, do not stand out at all on the paralysed leg.

If the patient sits on the edge of the bed with the legs hanging down, the dropping of the foot is very striking.

The toes also hang down, and this may be a marked symptom from the first. When the patient is standing there may be noticed some degree of flattening of the plantar arch.

Motor Disturbance.—No active movement is possible. In order to obviate any error of observation the patient's knee should be kept quite stiff while he attempts to carry out movements below that joint. The wounded man cannot stand on the heel or the toes of the paralysed foot. He cannot run.

The muscles of the back of the thigh are often affected. But as a rule it only amounts to a diminution of the power of flexing the knee, and this diminution affects rather the biceps cruris than the semitendinosus or the semimembranosus muscles.

When the wound is very high (in the buttock), complete paralysis of the three principal flexor muscles of the knee is observed.

Vasomotor, Secretory, and Thermal Disturbances.—Besides the immobility and dropping of the foot,

vasomotor symptoms demand attention. The foot and lower third or even the whole of the leg are swollen. Although on examining the patient in the morning after the night's rest only a moderate œdema may be noticed, a very considerable swelling will be present at night as the result of walking.

Whether definitely œdematous or simply infiltrated, the foot is generally of a purple colour, at any rate along its edges. The skin on the dorsum is fine and satin-like; in parts it may be covered with scales or crusts. Plantar hyperkeratosis is of frequent occurrence.

Sweating is noticeable for its absence on the dorsum of the foot as well as on the sole. Many of the wounded point out this symptom. They only perspire on the inner side of the foot in the area of the internal saphenous nerve, a branch of the anterior crural.

This congested, swollen foot with the delicate dry skin is generally warmer than its healthy fellow, at least in the first few months following the wound. This difference in temperature may amount to 2–3 degrees.

On the other hand, the calf of the wounded leg is often colder than the other, but this symptom is not so invariable.

The arterial tension taken at the level of the ankle by means of Pachon's sphygmomanometer is different from that of the healthy side; the pulsation appears earliest on the wounded side.

All these phenomena are, as Claude Bernard has demonstrated, in accord with a paralytic vasodilatation, due to the fact that the vasomotor and secretory fibres to the foot, carried by the sciatic, have been involved in the destructive lesion of that nerve.

These symptoms, noticeable during the earliest weeks of the wound, increase in intensity when the wounded man gets up and begins to walk. They gradually diminish as time goes on.

Sensory Disturbances.—Disturbances of subjective sensibility are usually absent. We have, however,

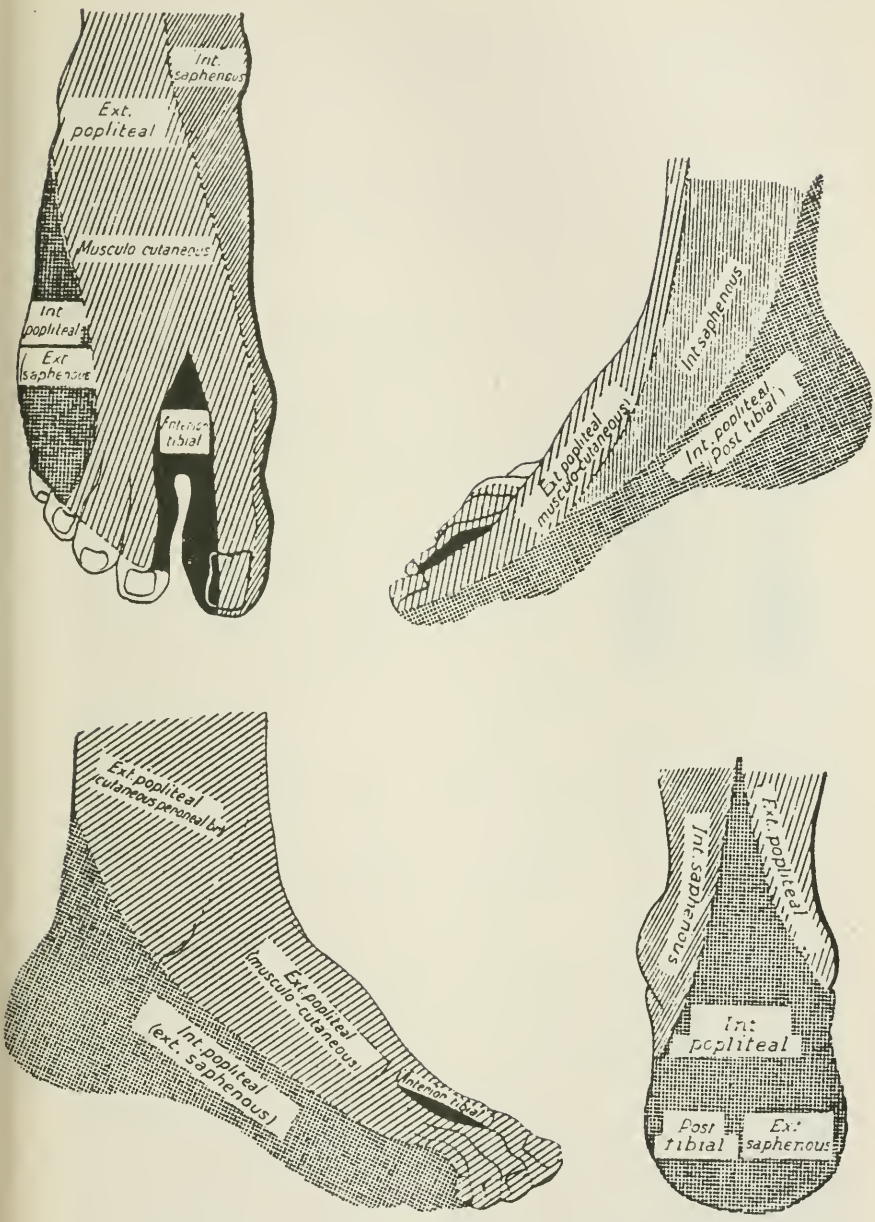


FIG. 73.—Peripheral sensory distribution of the foot.

met with patients who complained of tingling or twitching in the calf or in the foot, though the operation proved that the nerve was totally severed. These facts, which we cannot account for, are of rare occurrence.

The disturbances of objective sensibility consist in complete anæsthesia of the entire foot, with the exception of the inner border, the middle part of the internal plantar region, and the internal malleolus. This anæsthesia extends also on each side of the tendo Achillis, includes all the region of the external malleolus, and generally extends to the middle third of the external and posterior surface of the leg.

FIG. 74.—Distribution of sensory disturbance in the case of a grave lesion of the great sciatic nerve. —*Black:* complete anæsthesia to all forms of sensation. *Grey:* hypæsthesia.

Above and around this zone of complete anæsthesia there extends a region of hypæsthesia to prick, of more marked hypæsthesia to touch, and of anæsthesia to heat and cold.

And still further up, towards the knee, we sometimes find a zone of slight hypæsthesia to touch, with great hypæsthesia and delayed perception as regards medium temperatures.

The *sense of position* and *sensibility of bone* are abolished almost entirely in all the toes, in the sole, and in the back of the foot.

Some patients, however, distinctly feel movements in their tibio-tarsal articulation; they feel the wide, deep pressure of their foot as a vague shock. Nevertheless they do not feel the ground on which they walk, and they have the impression of having a wooden leg. *Sensibility to the vibrations of the tuning-fork* is often abolished over the external malleolus, and even along the shaft of the fibula; it may simply

be diminished over the internal malleolus, on the crest of the tibia, and on the body of the fibula.

Pressure on the external popliteal nerve at the neck of the fibula, on the internal popliteal in the popliteal space, on the posterior tibial nerve behind the internal malleolus, palpation of the trunk of the great sciatic nerve in the thigh below the wound—all these produce no perceptible pain, except in very rare cases.

Electrical Disturbances.—R. D. is complete in all the muscles of the leg and foot. It is frequently incomplete in the muscles on the back of the hip.

Faradic contractility is abolished in all the muscles if several weeks have elapsed since the wound.

The intensity of the galvanic current necessary to produce contraction, polar inversion, and longitudinal reaction vary from one muscle to another, but the essential phenomena of R. D. are always present.

Amyotrophy.—Atrophy of the leg cannot be perceived for some time, because of the œdema or infiltration of the calf. The size of the *hip* is diminished.

Reflexes.—The *knee-jerk* is present, and it is often more brisk than on the opposite side. The *ankle-jerk* as well as the *medio-plantar reflex* are always abolished; the *plantar cutaneous reflex*, when sought for on the inner border of the foot, sometimes produces a contraction of the fascia lata, but without any distinct movement of the foot.

Idiomuscular contractility of the muscles of the leg is generally increased in the earlier weeks; it diminishes in intensity in proportion as the atrophy increases. But from the first, percussion of the gastrocnemius produces much slower contraction than on the healthy side; this slowness also effects the relaxation of the muscular contraction.[1]

Percussion of the small muscles of the foot, such as the extensor brevis digitorum and interossei, and the internal aspect of the sole if the lesion has been

[1] Babinski, *Société de Neurologie de Paris*, March 4, 1915. André Thomas, *Société de Biologie*, Jan. 22, 1916.

in existence for any time, will not give rise to any contraction.[1] We have indicated elsewhere (p. 13) the principal points on which percussion should be performed.

EVOLUTION

In cases where the section has been complete, if no operation has been performed for suturing the nerve, at the end of several months the clinical symptoms become modified. The infiltration of the tissues grows less, atrophy appears and rapidly increases, and as the ligaments of the foot become distended it drops more. The dryness continues. There is sometimes plantar hyperkeratosis ; but more frequently atonic *ulceration* appears at the level of the heel, at the outer border of the foot, on the dorsal surface of the toes (especially if the dropping of the toes is accentuated), and also at the extremity of the great toe.

By reason of the anæsthesia of the foot the wounded do not feel knocks or the friction of badly fitting boots ; blisters form and develop into ulcers, which extend in depth and width under the influence of the original cause and walking.

These ulcerations take a long time to heal, but perfect cicatrisation is brought about by complete and prolonged rest in bed.

The *hair* does not always appear to be affected. In numerous cases it presents the same development as the healthy side. But if shaving takes place, as for an operation, the succeeding growth is very defective.

The *nails*, which are livid at the base and yellow at the extremity, are thick, friable, and transversely striated.

There is no change in the *anæsthesia* of the foot, but it may diminish very slightly in the leg.

Atrophy develops considerably ; the muscular development of the leg is very much reduced, and the diminution in the size of the hip is very perceptible.

[1] J. A. Sicard and Cantaloube, *loc. cit.*

Muscular percussion produces no contraction, or very little.

The *reaction of degeneration* results, at the end of several months, in complete faradic and galvanic inexcitability.

In spite of this loss of strength the infiltration of the tissues disappears. It is only the skin of the insensitive regions which is thickened.

The calf is always *cold*. The foot is often cold too, and in any case its degree of heat is lower than in the early stage. Its colour is purple, and the skin is dry.

RECOVERY

When regeneration of the nerve takes place, the appearance of painful symptoms, such as limping, twitching, and tingling in the calf and foot, precede recovery of motor power.

The restoration of objective sensibility is more retarded, and only established a short time before the return of movement.

Motor recovery comes almost suddenly. It is very easy to recognise, the sciatic nerve being the only one connected with the muscles of the leg and foot; any movement of the latter can only be attributed to a regeneration of that nerve.

Before the actual return of active movements the wounded man often says that when he desires to "command" his foot he feels as it were a current running through his muscles and suddenly blocked at the foot.

Then comes a day when he suddenly perceives, often after he has been massaged, that his foot can execute a certain movement in one position.

He tries to repeat this movement several times, but does not always succeed. He makes repeated efforts, and when he is just about to give up, suddenly the movement occurs. This movement is soon exhausted; the muscle is tired after two or three contractions.

The muscles which are the first to recover are, according to the individual, the *tibialis anticus*, the *peroneus longus* or the *gastrocnemius*; very much more rarely recovery begins in the tibialis posticus. In the course of serious lesions, or after suture, the flexor or extensor muscles of the toes are always the last to recover.

As the patient reports the return of a movement, he can indicate the most favourable position to obtain it. In order to move the tibialis anticus in most cases it is necessary to put the foot down flat, instead of letting it hang; the effort needed to raise the tendon is thus diminished. In this attitude, if the tendon does not always project during the efforts at movement, the substance of the muscle is felt to become hard on palpation immediately external to the shin.

Another manœuvre consists in raising the paralysed foot with the hand and flexing it forcibly on the leg; if the tibialis anticus contracts, it will keep the foot in this raised position for a few seconds.

To test the gastrocnemii the patient must be seated on his bed with the knee bent, the sole of the foot resting flat on the bed, then the calf must be grasped and palpated with both hands. If the patient tries to raise the heel, the sign of recovery will be the hardening of the muscles under the hands.

For the peronei the recumbent position is the best, the foot resting on the heel on the bed, the toes pointing upwards.

Mistakes may easily be avoided by keeping the patient's knee fixed to prevent the transmission of the movements of the thigh to the aponeurosis of the leg.

Recovery of movement makes very slow progress; movements take place in one muscle, and several weeks may pass before another recovers its motility.

As to the recuperation of motor *vigour*, it no doubt takes years in the case of a total section of the sciatic nerve.

As the motor symptoms improve, the sensory disturbances are modified. Zones of paræsthesia develop in areas where sensation was formerly dulled ; these paræsthetic phenomena are very numerous in sciatic paralysis. We shall study their characteristics in relation to the sensory restoration of the nerves.

Deep sensibility reappears. When great pressure is exercised on the foot, the patient has the impression of having the foot in a " vice " ; he feels passive movements impressed on it as though they came through a plaster casing.

Vasomotor symptoms disappear some time before the restoration of sensibility.

The return of electrical contractility, which may precede the return of voluntary movement, will be studied with more detail in the second volume of this work.

Atrophy does not visibly decrease till after the recovery of motility. Muscular tonicity is then rapidly re-established ; the dropping of the foot is less marked ; dangling disappears gradually, and this return of tonicity takes place more rapidly than motor recovery. A certain degree of muscular tonus reappears, however, before any sign of muscular contraction.

We have not yet found a case in which the ankle-jerk reappeared.

Signs of Recovery.—Sciatic paralysis may be considered cured when the wounded man can easily execute a " see-saw " movement—*i.e.* when standing with the feet together on the ground he must be able to balance himself alternately on the toes and on the heels (Pitres).

Another sign is being able to beat a measure in common time with the injured foot (Pitres).

B. INCOMPLETE PARALYSIS

It often happens that only the fibres of the external popliteal nerve are affected in the trunk of the great sciatic nerve. We shall study these cases when we deal with paralysis of the external popliteal.

In other cases the projectile has injured certain fibres of both of the popliteal nerves. We then get dissociated paralysis of the muscles supplied by these branches.

This dissociated paralysis is nearly always accompanied by painful phenomena when the lesion also affects the fibres of the internal popliteal, as we shall show when we discuss the painful forms of paralysis of the sciatic nerve.

Painful Forms of Lesions of the Sciatic Nerve

We have just seen how vasomotor, secretory, and sensory disturbances are accentuated in cases of serious lesion of the sciatic nerve in purely paralytic forms.

When the wound has injured the nerve without totally dividing it, the signs of recovery are definitely accompanied, more than in the case of any other nerve, by neuralgic pains and myalgia, which is not surprising considering the clinical history of this nerve (sciatic neuralgia), apart from any traumatism.

There are certain partial lesions which are accompanied, from the commencement, by painful phenomena exactly similar to those described in relation to the median nerve.

A wound of the great sciatic is only painful, it would seem, *when the fibres of the internal popliteal have been involved.* In the same way vasomotor, secretory, and caloric reactions are observed only in lesions where the fibres of this branch have been affected.

This is confirmed by what we observe in wounds situated below the bifurcation of the great sciatic. Only lesions of the internal popliteal or of its terminal branch, the posterior tibial, are painful.

The history of the wound is the same as that of purely paralytic lesions. Suppuration has not been more abundant. From the time of the wound there has been acute pain, but it was only a few days later that it took on its peculiar character.

The patient is confined to his bed. He is tortured by hyperæsthesia of the foot, particularly of the sole and toes. He keeps the knee half flexed, and in external rotation, to relax the nerve-trunk; he needs a cradle to keep off the weight and contact of the bed-clothes.

The foot is swollen and puffy, although pressure on the skin with the finger, for instance, cannot always cause pitting.

The skin is shiny, tense, and brownish-red in colour. Sweating is absent in the graver forms, but in slighter cases it is exaggerated.

From the first weeks the toes have a tendency to shrivel up and draw together. This deformity is most marked in the second phalanx of the great toe, and in the last two phalanges of the others.

The leg is very emaciated, and this emaciation extends to the thigh.

Motor Disturbances.—Movement is frequently quite impossible during the first weeks. Then motor recovery becomes perceptible, most often in the region of the internal popliteal (adduction of the foot; very slight flexion of the great toe or of all the toes at once).

In other cases where the fibres of the external popliteal are less injured, the movements controlled by that nerve are the first to be recovered.

Electrical Disturbances.—These vary in different cases, and always predominate in the muscles which are most affected.

Disturbances of Subjective Sensibility.—*Spontaneous pain* is compared to that of burning, smarting, and tearing; *it is localised in the sole of the foot*, particularly in its internal region, where reddish lines are often visible, and the skin has a punctate, granular, and pink appearance.

The pain often affects the *tips of the toes*, principally of the great toe, and the plantar region corresponding to the heads of the metatarsals.

The internal plantar region is sometimes the only part attacked, and is always the most affected.

In more complete forms all the toes are painful, including the dorsal surface of their third phalanx.

The pain occurs in paroxysms, and the entire foot becomes hyperæsthetic.

As in the case of the median nerve, the pain is influenced by a variety of causes—*e.g.* letting the limb hang down (the foot becoming red-brown), grazing or jarring it, noise, light, emotions, etc.

After vainly trying various soothing applications the patient has recourse to hot or cold water, wet compresses, and prolonged foot-baths.

The pain and irritation produced by touching any dry object extends to the whole body ; some patients damp their hands before touching anything at all.

Very slight pressure on the muscles, and especially on the trunk of the nerve below the wound, is very painful.

Disturbances of Objective Sensibility.—These are variable. There may be complete anæsthesia in the region of the external popliteal, and in a great part of the region of the internal popliteal. At other times there are simply anæsthesia to a superficial touch, and hyperæsthesia to pricking and heat throughout the sole of the foot.

In numerous cases there is a very clear distinction between the hyperæsthesia of the internal plantar region and the hypæsthesia of the external plantar region.

Reflexes.—The knee-jerk and gluteal reflex are very brisk. The *ankle-jerk* is always abolished.

Idiomuscular contractility is exaggerated, and this modification extends upwards to the gluteal muscles.

Vasomotor and Thermal Symptoms.—The temperature of the paralysed foot is higher than that of the healthy one. Arterial pressure is often affected, an increase of the maximum pressure (1 degree more than on the healthy side) being found.

EVOLUTION

Though time mitigates the pain, which generally disappears more quickly than in the case of the median

nerve, on the other hand it consolidates lesions such as muscular atrophy and articular ankylosis. The knee is flexed, the foot ankyloses with the leg, the extremity of the foot drops, the tendo Achillis retracts, the toes become flexed at the metatarso-phalangeal articulations, and the inner side of the foot turns inward to the sole. The foot becomes narrower and tapering (Léri). The pathogeny of these various deformities, and particularly that of clawing of the toes, is disputed ; they have been attributed to a retraction of the plantar aponeurosis or to a retraction of the tendons. In our opinion they are to be attributed to lesions of the articulations of the foot and toes rather than to any other cause.

FIG. 75. — Claw-like deformity of the toes following a lesion of the *great sciatic* (painful form).

The idiomuscular contractility remains exaggerated. The ankle-jerk is always abolished.[1]

The recovery of voluntary movement is slight, and takes a long time in serious cases.

Involuntary movements are sometimes observed when the ankyloses allow them to take place. These movements sometimes consist in an abduction of the foot followed by adduction, or in flexion of the great toe or all the toes. In one case we have observed a genuine movement of circumduction of the foot on the leg, although the patient could make no voluntary movement, and the R. D. was very marked in all the muscles of the leg and foot.

Radiography usually shows considerable decalcification of the bones of the foot.

[1] In one case we observed an inverted ankle-jerk : the percussion of the tendo Achillis was accompanied by a dorsal flexion of the foot and definite adduction.

When the wound is of old standing, the skin of the foot becomes brown and its temperature cold. In some parts branny desquamation or hyperkeratosis is to be observed. Ulcerations are sometimes seen in the anæsthetic areas.

Spontaneous pain gradually grows less. Dyæsthesia gradually diminishes in intensity, but continues to take the form of tactile hypæsthesia and hyperæsthesia to pricking.

Mistakes of interpretation and of localisation seemed to us to be very frequent in this form. Perception of heat and cold is particularly defective ; the patient is mistaken in quality. In some cases, when his foot is touched with a warm tube he says it is " warm," but a few instants later the sensation is divided : the " warm " continues to be felt, but, at the same time, an impression of cold is felt a little lower down. Weber's circles are enlarged. The patient often perceives only the point of the compass which is nearest the knee.

Disturbances of bone sensibility vary in different cases.

Painful Forms of Milder Degree

Besides the very painful forms, with pronounced motor and articular disturbance, there exist a multitude of slighter forms. In these the paralysis is not marked, or it is localised to a group of muscles. Ankylosis is not pronounced, and gradually yields to physio-therapeutic remedies. The less intense pains only affect the sole of the foot. Sweating is very abundant at first, infiltration of the foot is more or less developed, and electrical disturbance exists in proportion to the paralysis. But the ankle-jerk is always abolished, even when the gastrocnemius has retained its motility.

The pains grow less at the end of several months, and the secretory troubles disappear. But the involuntary movements, the aching in the calf, the pain caused by pressure on the nerve-trunk, the atrophy

and mechanical excitability of the muscles, persist for a long time.

In other cases the pains have no causalgic character at all, and do not affect the sole of the foot, but rather the tibio-tarsal articulation, or the outer surface of the calf. Walking then becomes possible in a very short time. But the ankle-jerk is always abolished, whatever may be the motor condition of the muscles of the calf.

Pathogeny.—In these painful forms of lesions of the sciatic nerve, the anatomical lesions are not serious: we have never met with a complete section of the nerve. It is generally a case of compression, crushing, or swelling of the nerve, which appears œdematous, congested, or, if the wound dates back several months, grey and indurated.

Suppuration or hæmorrhage was abundant in a few cases, while in others the patient neither lost blood nor suffered from sepsis.

To explain the pathogeny of these painful forms it is necessary to bear in mind the importance and the great number of the vasomotor, secretory, and other fibres which follow the trunk of the sciatic nerve, and which, after leaving the cord, accompany the posterior roots of the sacral plexus.

It is also necessary to bear in mind the region in which these painful phenomena develop: the sole of the foot, like the palm of the hand, contains a great number of specialised nervous corpuscles, some of which belong exclusively to the skin of these regions.

Lastly, as we stated with Dr Henry Meige in July 1915, we must take into account the vascularisation of the sciatic nerve, which has a very large artery (artery of the sciatic nerve, a branch of the sciatic), injury to which might be partly responsible for these altogether peculiar phenomena. Possibly there is an inflammation of the sympathetic fibres which accompany this artery. We are, however, inclined to admit an irritative lesion of the numerous sympathetic fibres following the trunk of the nerve, particularly a direct

EXPLANATION OF PLATE VII

A. *Radiography of the two hands of a patient suffering from a painful form of median nerve wound (causalgia), showing considerable decalcification of the bones.*

This decalcification predominates in the phalanges of the thumb and index finger, but it also affects those of the last three fingers as well as the heads of all the metacarpal bones.

The bones of the carpus are much less affected.

The decalcification is more marked in the neighbourhood of the inter-phalangeal and metacarpo-phalangeal articulations.

B. *Appearance of the leg and foot in a painful form of paralysis of the great sciatic nerve.*

Considerable atrophy of the muscles of the calf.

Development of the pilous system.

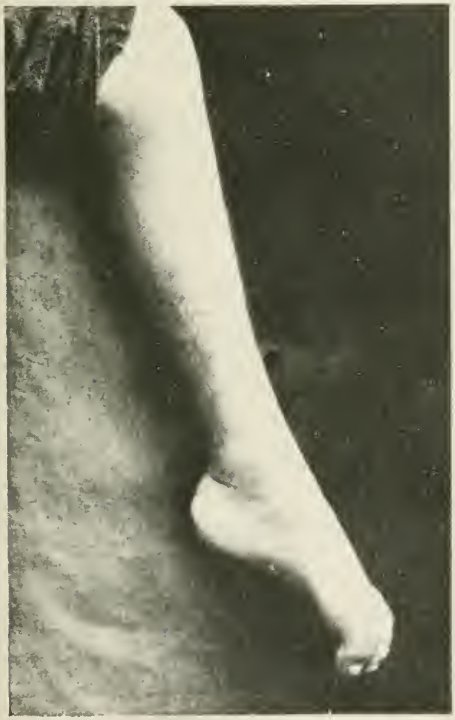
The foot is in extension (plantar flexion) on the leg through ankylosis of the tibio-tarsal articulation.

The toes, and particularly the great toe, are strongly flexed. This irreducible flexion is most marked in the last phalanx.

The inner border of the foot is also rolled inwards towards the plantar surface, and the whole foot is more contracted than the healthy one.

C. *The same foot seen on its dorsal surface. The "rolled-in" appearance of the internal edge comes out very distinctly.*

A



B

C

PLATE VII.—Nerve lesions (clinical forms).

lesion of those centrifugal fibres that spread out and form the organ of Timofeew, round the sensory corpuscles of the skin, and round their central myelinated fibre (page 84).

What is the nature of this lesion ? As in the case of the median nerve, we consider that it is a case of a chronic interstitial inflammation, as might be concluded from the congested, œdematous appearance of the nerve in recent lesions of this description, and from the induration and shrunken greyish aspect of the nerve in older lesions.

Paralysis of the External Popliteal Nerve

A lesion affecting the external popliteal nerve in the popliteal space below the bifurcation of the great sciatic nerve produces the same paralytic and sensory disturbances as a wound in the fibres of the external part of the great sciatic trunk. The differences that may exist between the two clinical forms are very slight, and will be indicated in the course of this description.

A. COMPLETE PARALYSIS

The great majority of wounds of the external popliteal cause immediate and extensive paralysis of the muscles of the antero-external region of the leg and of the peroneus longus and brevis.

The external popliteal nerve behaves in this respect exactly like the musculo-spiral nerve.

The phenomena which immediately follow the wound show nothing characteristic—pain like that from the lash of a whip, a sensation like that of the leg being torn away, etc. The only constant event is the immediate foot-drop.

Appearance and Gait.—At the end of four to six weeks, if there has been no serious infection or considerable fracture of the bones, the patient begins to walk.

He walks with a steppage gait, raising the knee high, the point of the foot dropping and in adduction

during the forward step; the foot is planted heavily on the ground, and the plantar arch is somewhat flattened.[1]

Some men show an exaggerated degree of steppage gait, flinging out the foot, so much so that they prefer to walk with crutches until they are made to wear a prosthetic apparatus correcting this action of the foot.

Cases of this kind tend partly to verify the witticism of Duchenne (of Boulogne): "It is better to lose all the motor muscles of the foot and leg than to keep some of them only."

When the patient is sitting the foot-drop is accentuated, sometimes even more so than in a case of total paralysis of the sciatic nerve.

A slight drop of the first phalanx of all the toes is observed. When the patient is lying down with the leg extended, it will be found that the plantar surface of the foot turns in, and that the foot dangles when sudden movements are imparted to the leg.

If the paralysis dates back some time, a slackening and distension of the ligaments of the ankle are observed. Paralysis of the extensor longus, which is normally an active and very effective ligament of the medio-tarsal articulation, produces a flexion of the foot at this point which increases its dangling. It is not uncommon to find in such a case *a dorsal tumour of the tarsus*, which is usually due to the more or less pronounced projection of the heads of the astragalus and scaphoid bones, and to the thickening of the sheaths of the extensor tendons.

Motor Disturbances.—To examine the condition of voluntary movement it is necessary to render the knee of the patient immovable, and to palpate the muscles and the different tendons at the ankle attentively, so as not to mistake the twitchings of the

[1] This flattening is not very pronounced, probably owing to the associated paralysis of the tibialis anticus, which in the normal state draws the first metatarsal in an opposite direction from the action of the peroneus longus. When the peroneus longus is paralysed, the predominant action of the tibialis anticus causes the complete flattening of the plantar arch.

aponeurosis of the leg caused by the muscles of the thigh, or the contractions of the other muscles of the leg, for active movements of the antero-external group of muscles.

Under these conditions no active movement of dorsal flexion of the foot (tibialis anticus) is possible.

The movements of adduction executed by the foot during this effort to raise the foot are due to the tibialis posticus, which projects behind the internal malleolus.

Extension of the proximal phalanges of the toes is impossible, the *extensor longus digitorum*, the *extensor proprius hallucis*, and the *extensor brevis digitorum* being paralysed. But the contraction of the interosseous tendons, which in the foot, as in the hand, extend the last two phalanges, might make one think that there was an extension of the toes. During these efforts at extension, however, the first phalanx is not affected, and the extensor tendons, so prominent in the normal state beneath the skin of the back of the foot, do not project at all.

Genuine abduction of the foot cannot be accomplished (paralysis of the peroneus longus and brevis); when the patient is ordered to turn his foot outward he forcibly contracts the gastrocnemius and soleus, and lowers the point of the foot. Examination of the peroneal tendons behind the external malleolus reveals no appreciable contraction. Owing to the weakness of the muscles which we have just mentioned, the patient cannot walk on the heel of the paralysed side, he cannot run because of the foot-drop, and though he can stand on tiptoe, he experiences some difficulty in keeping his balance.

Atrophy of the muscles of the antero-external group, hardly perceptible at first, increases as time goes on.

Electrical Disturbances.—As in all complete and serious paralysis, electro-diagnosis shows loss of faradic and galvanic excitability of the nerve and a reaction of degeneration in the paralysed muscles.

When the lesion is of old standing and the muscular

impairment advanced, the longitudinal reaction is more active than the reaction at the points of election; then the loss of faradic and galvanic excitability of the muscles, and the rapid diffusion of the current into the gastrocnemii, the tibialis posticus, and the flexors succeed the initial reaction of degeneration.

Subjective Sensory Disturbances.—Painful phenomena are rare in the case of lesions of the external popliteal nerve. When they exist they consist of aching pains in the upper part of the outer surface of the leg, in which case they are due to lesions in the bones (fracture or fissure of the head of the fibula) or ligaments.

In other cases the pain is more intense, and is seated in the malleolar region, radiating towards the external border of the foot, and in rarer cases towards its dorsal surface.

In the great majority of these cases we found an abolition or a great diminution of the ankle-jerk.

The lesion has then affected the trunk of the sciatic nerve above its bifurcation, with serious involvement of the fibres of the external popliteal, and it has also caused some irritation of the fibres of the internal popliteal, hence the existence of the pain and the loss of the ankle-jerk.[1]

When the lesion is seated lower down in the popliteal space, the pain is due to the partial embedding of the internal popliteal nerve in the fibrous cicatricial tissue of the wound.[2]

Objective Sensory Disturbances.—Disturbances of *objective sensibility* occur on the dorsal surface of the foot, in the area of the musculo-cutaneous, and on the outer surface of the leg, in the areas of the cutaneous peroneal, and communicans peronei nerve regions.

In the foot diminution of sensibility reaches to the

[1] In this case the gastrocnemii show slight electrical changes.

[2] We have observed one case of complete section of the external popliteal nerve with pain in the ankle and loss of the ankle-jerk. At the operation the upper end of the nerve was found to be adherent to the internal popliteal, which was quite unaffected.

base of the toes, and extends inwards to the inner border of the foot, and outwards to the fifth metatarsal.

On the external surface of the leg this disturbance of sensation rises to different heights, but it does not generally extend further than the middle third, and does not reach the shin.

Round the zone of anæsthesia we generally find a band of hypæsthesia of greater or less extent. Pressure on the trunk of the nerve at the neck of the fibula does not cause pain, unless there are local lesions of the bone.

Reflexes. — The knee- and ankle-jerks are present. We have seen cases in which the latter was absent. The plantar cutaneous reflex is always flexor.

Idiomuscular contraction is exaggerated in the earlier stages in the large muscles of the antero-external region and in the peronei.

It diminishes in intensity, and gets rather slower when the paralytic lesion is several months old. This slow muscular contraction is best seen in the extensor brevis digitorum in the earlier weeks (Sicard); it is not long in disappearing completely as the muscular atrophy progresses.

FIG. 76. — Zone of sensory changes of the dorsum of the foot in a case of lesion of the external popliteal nerve.

FIG. 77.—Distribution of sensory disturbance in lesions of the external popliteal nerve. The black area represents anæsthesia, the dotted area hypæsthesia.

Vasomotor Disturbances —These are not important. In rare cases there is a purple discolouration of the foot,

which is colder than the one on the opposite side. Œdema can never be detected.

The skin is normal, and there is no important change in the nails or in the pilous system. Desquamation of the epidermis with diminution or disappearance of sweating may sometimes be observed in the first two dorsal interosseous spaces.

The subcutaneous tissue is slightly infiltrated on the external surface of the leg, as can be found by pinching the skin.

Recovery.—Restoration (spontaneous or after suture of the divided nerve) takes place only after several months. The first favourable sign is the appearance of subjective disturbance of sensibility, such as *pain on pressure of the nerve-trunk and on pinching the skin in the sensory region of the nerve.*

Motor recovery returns more slowly ; at the same time the sensory impairment begins to clear up.

The tibialis anticus is generally the first to recover its motility. This restoration can be detected by feeling the body of the muscle, which hardens when the effort is made, and by noticing the movement of its large tendon on the front of the ankle.

At other times the peronei are the first to recover, while the extensor longus digitorum and extensor proprius hallucis always recover their motility last of all. When the ligamentous distension is very pronounced, the appearance of the first voluntary movements does not always modify the attitude of the limb. The patient continues his steppage gait as in the early weeks, the point of the foot continuing to drop.

But when once the muscles are in the way of recovery the improvement in their tonicity becomes evident, atrophy disappears more quickly, and this progress is more rapid than that of voluntary movement.

Complete recovery does not take place for a very long period ; in some cases the return to a normal state may take years. In fact, we have not yet, after a period of twenty months, met with a case of complete

recovery of a serious lesion of the external popliteal nerve.

Signs of Recovery.—A patient of this sort may be considered to have completely recovered when he can beat a measure in common time, and when he can support himself alternately on the heel and on the point of the foot (Pitres).

B. DISSOCIATED PARALYSIS

Cases of incomplete and dissociated paralysis are met with, but not very frequently; they generally occur after wounds of the leg, in which the musculo-cutaneous alone or the anterior tibial have been involved. But lesion of the nerve above its bifurcation may occasion sensory and paralytic disturbances confined to a limited area.

When the paralysis predominates in the tibialis anticus and the extensors of the toes the foot hangs down, but is not adducted. The plantar arch is normal, but abduction of the foot is often more marked than in complete paralysis.

Sensory disturbances then occur only in the first dorsal interosseous space.

The study of operative results, and particularly the direct electrization of injured and healthy nerves, shows that in the trunk of the external popliteal nerve in the popliteal space the motor fibres are arranged in the following manner :—

On the *antero-external* surface of the nerve :—the fibres supplying the *extensor longus digitorum.*

On the *antero-internal surface :*—the *tibialis anticus.*

On the external surface :—the *extensor proprius hallucis.*

On the posterior surface :—the *peroneus longus and brevis.*

Paralysis of the Internal Popliteal Nerve

Complete and isolated paralysis of the internal popliteal by direct lesion of the nerve in the popliteal

space, or by injury to the internal fibres of the great sciatic trunk, is rather rare. What we meet with most often is partial or dissociated paralysis of this nerve, accompanied by more or less complete paralysis of the external popliteal nerve. The pain being in these cases a constant phenomenon, the study of dissociated paralysis is included in that of the painful forms of the sciatic nerve described above. In the wounds of the internal popliteal which we have met with, we have not found a case of complete section of the nerve.

We will therefore describe a form of complete paralysis caused by serious lesion of the nerve but without complete division.

Appearance and Gait.—The wounded man appears with one foot hanging down and œdematous and the toes swollen, having their first phalanx extended and the two others flexed. The projection of the tendo Achillis is absent.

Œdematous infiltration of the foot extends to the leg, reaching almost up to the knee.

The colour of the skin is purple-red ; in the plantar region areas of hyperkeratosis are often present; sweating is diminished. The deformity of the hanging foot with its lowered heel and raised metatarsus is generally accompanied by stiffness of the tibio-tarsal articulation, which renders the reduction of this attitude difficult or impossible. Hence there is considerable difficulty in walking, and the wounded men are obliged to use crutches.

Motor Disturbances.—No movement for lowering the foot can be executed; the *gastrocnemii* muscles are absolutely flabby, and the tendo Achillis does not stand out.

Frank adduction of the foot (tibialis posticus) is impossible ; it is always accompanied by elevation of the foot, and it is then due to the contraction of the tibialis anticus.

Flexion (flexors of the toes) and separation of the toes (interossei) are abolished, and no muscle or tendon

moves in the sole of the foot. Sometimes, when the movements of the extensor muscles of the toes are well maintained, there is a semblance of flexion of the toes analogous to the phenomenon which we observed in the hand, caused by the sudden relaxation of the extensor tendons after an active hyperextension of the first phalanges.

Finally, when the fibres of the nerve have been injured within the great sciatic trunk, in the gluteal region, a paralysis, which is sometimes complete, of the flexor muscles of the leg may be observed.

Electrical Disturbances.—These are not always easy to estimate when the œdematous infiltration is very considerable. Generally the reaction of degeneration is present in the muscles of the calf and of the sole of the foot.

Reflex Disturbances.—The ankle-jerk is abolished; idiomuscular contraction is often exaggerated at first, but it soon diminishes in intensity and becomes sluggish.

Sensory Disturbances.—The disturbances of *objective sensibility* occur in the sole of the foot (except on the inner border), on the plantar surface of the toes, sometimes also on the dorsal surface of their last phalanx, on the external border of the foot, where they encroach more or less on the dorsal surface, and in the calcaneal region, where they extend above the insertion of the tendo Achillis.

Sensibility of bone is abolished throughout the sole of the foot, and the sense of position is diminished in the toes.

Painful phenomena of a neuralgic character occurring in the calf and ankle, or definitely causalgic localised in the plantar region, are very frequent in cases of incomplete lesions of the nerve, as we have often pointed out.

Trophic Disturbances.—These are not very pronounced. The nails, as in total lesions of the sciatic nerve, may become more friable, yellowish or livid. But the loss of the nails, the bluish colour of the

skin, large sloughs or gangrene ending in partial amputation of one or several toes, are hardly ever met with, unless an arterial lesion is associated with the nervous lesion, as in certain wounds in the popliteal space.

Paralysis of the Posterior Tibial Nerve.—When the projectile (bullet or shell fragment) has penetrated the calf, a *lesion of the posterior tibial nerve* may be found with paralysis of all the muscles of the sole of the foot, or dissociated paralysis of the internal plantar muscles, and pain of a causalgic nature, profuse sweating, and the pink colour characteristic of the skin in the sensory area of this branch only.

The researches of Pierre Marie, Henry Meige, and Gosset show that from the motor point of view the fibres of the internal popliteal nerve seem to be arranged in the following manner: in the *postero-internal* region of the trunk the fibres of the *flexors of the toes*, and on its *posterior* surface those of the *gastrocnemius* and *soleus*. The fibres of the *tibialis posticus* appear to occupy the *antero-internal* region.

M. and Mme. Dejerine have published two cases of partial paralysis of the internal popliteal nerve, which are interesting on account of the effort made by the authors to localise by clinical examination alone the position of the motor and sensory fibres in the interior of the nerve-trunk.

In the first case the lesion occupied the fifth internal division of the great sciatic nerve in the gluteal region. The most marked disturbances were found in the territory of the external saphenous nerve and the interosseous nerves. The nerves to the gastrocnemius and soleus and the plantars were less affected, while the other branches of the internal popliteal nerve were almost intact.

In the second case, where the lesion affected the external third of the internal popliteal nerve immediately below its origin, the paralysis and sensory disturbance predominated in the plantar nerves (particularly in the superficial branch of the external plantar

nerve), the calcaneous branches, the flexor nerves of the toes, and in a slighter degree in the nerve to the tibialis posticus.

The nerves to the gastrocnemius and soleus, the deep branch of the external plantar, and the external saphenous nerve were more or less intact.

From these two cases the authors draw the following conclusions regarding the intra-truncal nervous localisation :—

The internal saphenous nerve seems to be closely connected with the most internal part of the sciatic nerve both in the gluteal region and in the popliteal space. Following it therefore from the interior to the exterior, the different fibres may be localised as follows : the deep branch of the external plantar nerve, the internal plantar nerve, the branch to the gastrocnemius and soleus, the nerve to the tibialis posticus, the nerves of the flexors, the superficial branch of the external plantar nerve, and the calcaneous branches.

CHAPTER X

THE LUMBO-SACRAL PLEXUS

In the lower limb, apart from the great sciatic nerve, the remaining nerves (anterior, crural, obturator, etc.) are very rarely affected. We shall add a few remarks on this subject at the end of this chapter.

The lumbar and the sacral plexus are also rarely attacked separately. The most usual occurrence is a bilateral lesion of the "lumbo-sacral" plexus (lumbar roots and sacral roots), in consequence of the projectile having wounded the "cauda equina"[1] in its intra-spinal course, inside or outside of the dural sac.

As a matter of fact, lesions of the lumbar and sacral regions seem more frequent, and in all cases more often accompanied by nervous lesions than wounds of the anterior and internal surface of the thigh.

ANATOMY

The **lumbar plexus** is formed by the collection of anastomotic loops given off by the anterior branches of the first three lumbar nerves and a part of the fourth.

The rest of the latter unites with the fifth, takes the name of the *lumbo-sacral cord*, and joins the sacral roots to form the *sacral plexus*.

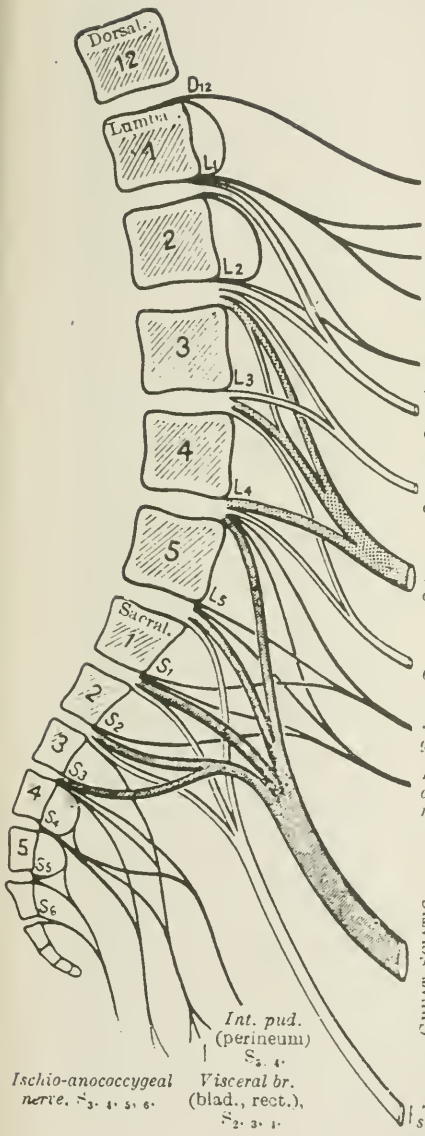
The sacral plexus results from the fusion of the lumbo-sacral cord with the first three sacrals.

The *pudendal plexus* is formed of nerves derived from the anterior branches of the third and fourth sacral nerves.

Unlike the brachial plexus, in which the connections of the

[1] The *cauda equina* is formed by the last lumbar nerves and the sacral nerves.

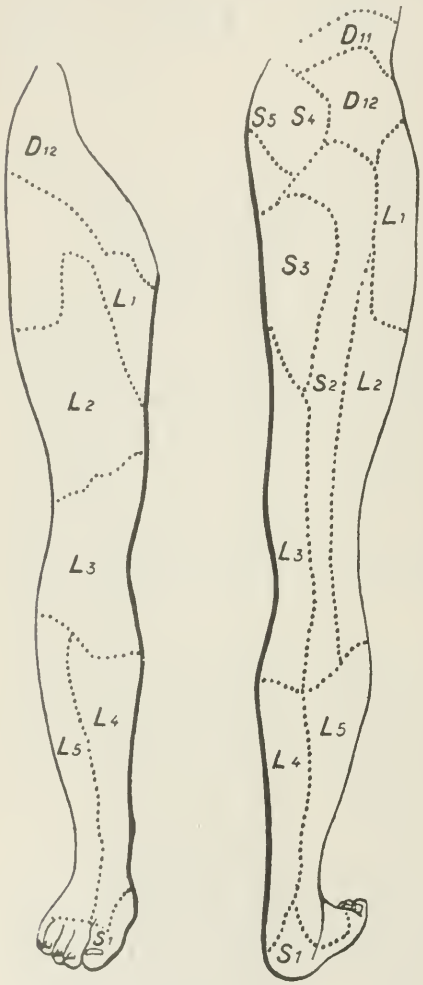
LUMBO-SACRAL PLEXUS



NERVES.	MUSCLES.	ROOTS.
12th interc. N.	Intercostal.	D_{12}.
N. to quad. lumborum.	Quadratus lumborum.	D_{12}, L_1.
Ilio-hypo.	External and internal oblique.	D_{12}, L_1.
Ilio-inguinal.	Tranversalis.	
Nerve to psoas.	Psoas.	L_1, L_2.
Genito-crural.	Tranversalis. Iliacus.	L_1, L_2.
External cutaneous.		L_2, L_3.
Anterior crural.	Iliacus. Psoas. Pectineus. Quad. femoris. Sartor. Adductor longus.	L_2, L_3, L_4.
Obturator.	Adductors. Obturator ext. Gracilis.	L_2, L_3, L_4.
Superior gluteal.	Glutei. Tensor fasciæ femoris.	L_4, L_5, S_1.
Pelvi-trochanteric muscles.	Obturator inter. Gemelli. Pyr. Quad. fem.	L_4, L_5, S_1, S_2.
GREAT SCIATIC. *External poplit.*	Adductor longus. Biceps. Semitendinosus, semimembranosus. Tib. ant. Ext. long. and brevis dig. Peron. tert. Peroneus longus and brevis.	L_4, L_5, S_1, S_2.
GREAT SCIATIC. *Internal poplit.*	Soleus. Gastroc. Pop. Plant. Tib. post. Flex. long. and brevis dig. Plantar muscles.	L_4, L_5, S_1, S_2.
Small sciatic.	Gluteus maximus.	L_5, S_1, S_2.

PLATE VIII.

primary and secondary trunks and their branches constitute a nervous puzzle which it is necessary to describe, the arrangement of the lumbar and sacral roots is very simple. Plate VIII. will show their anatomy better than a long description.



(Anterior aspect.) (Posterior aspect.)

FIG. 78.—Sensory radicular distribution of the lower limb. (after Head.)

Collateral Branches of the Lumbar Plexus.—(*a*) *Short branches.*—1. Nerves to the *quadratus lumborum* (L^1). 2. Nerves to the *psoas* (L^2). 3. Nerves to the *psoas parvus.*

(*b*) *Long Branches.* — 1. The *ilio-hypogastric* nerve (D^{12}, L^1) sends a *deep cutaneous* branch to the skin of the buttock, and divides (at the anterior part of the iliac crest) into two branches: (*a*) *abdominal*, for the muscles of the abdomen and the skin of that region; (*b*) *genital*, for the skin of the pubis and scrotum.

2. The *ilio-inguinal* (D^{12}, L^1), more slender than the preceding, to which it is closely related, presents an analogous arrangement and distribution.

3. The *genito-crural nerve* (L^1, L^2) is divided into a *genital branch*, which traverses the whole extent of the inguinal canal and supplies the skin of the scrotum, and a *crural branch* for the skin of the anterior and superior surface of the thigh.

4. The *external cutaneous* (L^2, L^3) leaves the pelvis through the unnamed notch between the anterior and inferior iliac spines. Two fingers' breadths below it divides into a *gluteal branch* for the skin

of the buttock, and a *femoral branch* which descends to the knee, covering with its ramifications the skin of the antero-external region of the thigh.

Terminal Branches of the Lumbar Plexus.—These are two in number, the obturator and the anterior crural.

I. **The Obturator Nerve** (L^2, L^3, L^4) passes into the pelvis on the inner side of the psoas muscle, crosses the sacro-iliac articulation, and, escaping by the sub-pubic groove, reaches the antero-internal part of the hip.

At this point it furnishes a collateral branch for the *obturator externus* muscle, and divides into two terminal branches :—

(*A*) A *superficial branch*, which, on arriving between the adductor longus and brevis, ends in four branches, three motor ones for the *gracilis*, *adductor longus*, and *adductor brevis*, and a *cutaneous branch* for the skin of the supero-internal region of the thigh.

(*B*) A *deep branch*, whose principal ramifications are motor, and supply the adductor magnus.

II. **The Anterior Crural Nerve** (L^2, L^3, L^4).—This, the largest nerve of the lumbar plexus, first passes down in the pelvis under the aponeurosis of the ilio-psoas in the groove formed between the psoas and iliacus, and then appears under Poupart's ligament, immediately external to the femoral vessels.

At this point, or a little below it, the anterior crural nerve divides into its terminal branches.

The anterior crural nerve supplies, as collateral branches, motor filaments to the psoas, the iliacus, and the pectineus.

The terminal branches are four in number :—

(*a*) The *external musculo-cutaneous nerve*, the most external branch of the superficial division of the anterior crural, furnishes muscular branches to the *sartorius* and *cutaneous* branches to the anterior region of the thigh.

(*b*) The *internal musculo-cutaneous nerve* supplies muscular branches to the pectineus and the adductor longus, and cutaneous filaments to the antero-internal region of the thigh.

(*c*) The *nerve to the quadriceps femoris*, the deepest of the terminal branches of the anterior crural, divides into four branches for the four divisions of the muscle—the *rectus*, *vastus externus*, *vastus internus*, and *crureus*.

(*d*) The *internal saphenous nerve*, exclusively sensory, extends from the fold of the groin to the internal border of the foot. It is the longest branch of the anterior crural, of which it appears to be the continuation. Situated at its commencement, external to the sheath of the femoral vessels, it penetrates the sheath at about the middle third of the thigh, and then passes directly in front of the femoral artery, in the same compartment with it. It leaves Hunter's canal shortly before the passage of the femoral artery

into the ring of the adductor brevis, perforates the aponeurosis of the leg near the internal tuberosity of the tibia, becoming subcutaneous in relationship with the internal saphenous vein, and divides into its two terminal branches.

Among its *collateral* branches we may note the *cutaneous femoral branch*, which begins at about the middle of the thigh and is distributed to the postero-internal region of the thigh and knee, and the *cutaneous tibial branch*, the terminal branches of which supply the skin of the internal region of the calf.

The *terminal* branches are two in number :—

1. An interior or *patellar* branch arises at about the level of the internal condyle of the femur and proceeds towards the anterior surface of the knee, describing a curve with an upper concavity surrounding the inferior extremity of the patella. It supplies the skin covering the region of the knee.

2. An inferior or *crural* branch follows the same course as the internal saphenous vein, its anterior branch supplying the internal border of the foot, and its posterior branch the skin over the internal malleolus.

Sacral Plexus.—The sacral plexus is formed by the fusion of the anterior branches of the first sacral nerves (S^1, S^2, S^3, S^4) joined by the lumbo-sacral cord (L^4, L^5), and below by a slender branch of the fifth sacral nerve.[1]

The sacral plexus as a whole has the form of a triangle, the base of which corresponds to the anterior surface of the sacrum, while its apex is situated a little above the spine of the ischium.

The Great Sciatic Nerve, which is derived from the fourth and fifth lumbars (through the medium of the lumbo-sacral trunk), the first, second, and third sacral nerves, and partly from the fourth, comprises thus almost the whole of its fibres, and constitutes its only *terminal* branch.

The *collateral branches* of the sacral plexus are ten in number. They may be divided into anterior or intra-pelvic, and posterior or extra-pelvic branches.

(*A*) The **anterior branches** are five in number :—

1. The slender *visceral branches* are given off from the last sacral nerves, and their destination is the pelvic viscera, the bladder, rectum, etc.

2. The *nerve to the obturator internus* (L^5, S^1).

3. The *nerve to the levator ani* (S^3, S^4).

4. The *hæmorrhoidal or anal nerve* (S^3, S^4), which supplies the soft parts of the perineal region and the external sphincter of the anus.

5. The *internal pudic nerve* (S^2, S^3, S^4), of considerable size, divides

[1] Poirier-Charpy-Cunéo, *Abrégé d'Anatomie*, vol. ii.

into two terminal branches—the perineal nerve and the dorsal nerve of the penis.

(*a*) The *perineal nerve*, which supplies the greater part of the muscles of the *perineum* and the skin of the scrotum in man.

(*b*) The *dorsal nerve of the penis* supplies the striated muscle of the urethra and the skin of the lateral parts of the penis.

(*B*) The **posterior branches,** also five in number, are more important; they are distributed among the muscles of the pelvic girdle and to the skin of the posterior surface of the thigh.

1. The *nerve to the pyramidalis* (S^1, S^2).
2. The *nerve to the gemellus superior* (L^5, S^2).
3. The *nerve to the gemellus inferior and quadratus femoris* (L^4, L^5, S^1).
4. The *superior gluteal nerve* (L^5, S^1) supplies the *gluteus medius*, the *gluteus minimus*, and the *tensor fasciæ femoris*.
5. The *inferior gluteal or small sciatic nerve* (L^5, S^1, S^2) constitutes the largest collateral branch of the sacral plexus.

It leaves the pelvis below the pyramidalis muscle, with the great sciatic nerve, behind which it is placed, and of which it seems to be the accessory. It soon divides into two terminal branches, one muscular and the other cutaneous.

(*a*) The *muscular branch* supplies the *gluteus maximus*, which it penetrates on its anterior surface.

(*b*) The *cutaneous branch* (*posterior cutaneous nerve of the thigh*) descends vertically towards the posterior surface of the thigh, and divides in the popliteal region into its terminal branches.

The posterior cutaneous nerve is sensory; it gives off the following collateral branches:—

(*a*) *Gluteal branches* to the skin of the gluteal region.

(*b*) *Perineal or genital branches* to the femoro-perineal fold, perineum, and the scrotum.

(*c*) *Femoral branches* to the posterior surface of the thigh.

Its *terminal branches* are distributed to the posterior and superior surface of the calf.

PHYSIOLOGY OF THE MUSCLES OF THE HIP

I. *Gluteus maximus.*—This is above all a *very powerful extensor* of the thigh on the pelvis, and *vice versa*. It causes outward rotation of the thigh without force, and is not at all an abductor.

In walking on flat ground and standing up the glutei do not play the important part that has been attributed to them.

A man in whom the glutei are the only muscles paralysed can stand without fatigue and walk without apparent lameness on a horizontal surface.

But he can hardly raise himself if he is seated, and he has the

greatest difficulty in going upstairs, walking on rising ground, jumping, running, etc.

In short, the principal function of the gluteus maximus muscle is to extend the thigh powerfully on the pelvis and the pelvis on the thigh at the moment when they are flexed on one another, as in going upstairs.

Clinically the weakness of this muscle can be realised by making the patient go up one or two stairs ; the observer should meanwhile carefully palpate the muscles to note its contractions. A still more simple plan is to make the patient lie flat on his stomach on the bed and to order him to close his buttocks.

The muscle of the affected side will remain flabby, while on the sound side it hardens and draws nearer the median line.

2. *Gluteus medius.*—Contraction of the whole muscle produces *direct abduction of the thigh.*

Isolated contraction of the anterior fibres produces internal rotation with abduction and slight flexion, and that of the posterior fibres causes external rotation with abduction and extension.

Having its fixed point on the femur, it plays an important part in the fixation of the pelvis, especially when the hip is flexed, or second stage of walking.

In cases of paralysis of the gluteus medius and of the gluteus minimus, whose functions are identical, there is an inclination of the pelvis to the opposite side when the patient stands on the affected limb.

To test the force of resistance of the gluteus medius, which is pre-eminently the abductor muscle of the thigh, the lower limb which is being examined must be drawn apart, and the patient must be ordered to retain this position and to oppose any movement which would tend to bring the two thighs together.

3. *Gluteus minimus.*—This muscle has the same actions as the gluteus medius.

4. The *tensor fasciæ femoris* is a flexor and internal rotator of the thigh. It also makes tense the external part of the femoral aponeurosis.

5. The *pyramidalis, gemelli, quadratus femoris, obturator internus,* and *obturator externus,* also called pelvi-trochanteric muscles, are all *external rotators* of the thigh, and in this respect antagonists of the gluteus medius and gluteus minimus.

To test their power of resistance the lower limb must be placed in external rotation, the foot pointing outwards, and the patient must be told to oppose any movement which would tend to turn his thigh inwards.

6. *Ilio-psoas.*—The ilio-psoas muscle powerfully flexes the thigh on the pelvis, giving it at the same time a slight outward rotation ; while the tensor fasciæ femoris, another flexor of the thigh, produces an inward rotation.

The power of resistance of the ilio-psoas is explored by flexing the patient's thigh on the pelvis, and by making him oppose, as energetically as possible, any attempt at extension of the thigh.

Examination of this muscle should always be made by comparing it with the sound side, and the doctor should feel the anterior surface of the thigh only with the tips of his fingers, the force of resistance of the flexors of the thigh being normally inferior to that of the extensors, the abductors, or the adductors of this part.

7. The *three adductor muscles* (adductor magnus, adductor longus, and adductor brevis), the gracilis, and the pectineus are the adductor muscles of the thigh—that is to say, they draw it powerfully towards the axis of the body.

To ascertain their power of resistance, which in the normal state is very great, the patient being in the recumbent position should be ordered to draw the thighs forcibly together, while the observer, with his two hands between the patient's thighs, opposes the latter's effort with all his might.

In the case of wounded men whose adductors are atrophied, instead of the leg swinging directly from behind forwards during the second part of the walking step, it is moved rather obliquely outwards and forwards.

8. *Quadriceps femoris.*—The four chief parts of this muscle (rectus, vastus externus, vastus internus, and crureus) by contracting simultaneously cause *extension of the leg on the thigh.* Their action is very powerful.

The vastus externus and vastus internus parts, when they are made to contract separately by a strong faradic current, each extend the leg on the thigh by drawing the patella obliquely, the former outwards and upwards, the latter inwards and upwards.

During their simultaneous contraction the patella is drawn directly upwards. The power of extension of the vastus internus is greater than that of the vastus externus.

The gracilis, besides extending the leg, causes a flexion of the thigh on the pelvis.

The power of resistance of the quadriceps is very great in the normal state. To ascertain this the patient should be ordered to extend the leg energetically, and to oppose all efforts at flexion made by the observer.

9. The *sartorius flexes the leg on the thigh and the thigh on the pelvis.* It concurs with the other muscles in the movement of swinging from behind forwards in the second stage of walking.

It does not exert any movement of abduction on the lower limb, and does not, as has been wrongly stated, make it assume the ordinary attitude of the tailor.

To sum up, *flexion of the thigh on the pelvis* is carried out by the

ilio-psoas, tensor fasciæ femoris, sartorius, and *gracilis,* the first being the most important.

Extension of the thigh on the pelvis is the work of the *gluteus maximus.*

Abduction of the thigh is due to the *gluteus medius* and *gluteus minimus,* while *adduction* is due to the *adductors, pectineus,* and *gracilis.*

External rotation of the thigh is executed by the pelvi-trochanteric muscles, and inward rotation by the gluteus medius and gluteus minimus.

CLINICAL STUDY

Wounds of the *cauda equina* are characterised by the fact that they involve the roots unequally ; paralysis and disturbance of sensibility do not affect both lower limbs in the same way.

Sphincter troubles are frequent ; they consist in a retention of fæces and of urine, which is soon followed by incontinence. Disorders of defæcation improve more frequently than those of micturition, which seldom clear up entirely.

Sphincter disturbances are often accompanied by the development of bedsores, which are sometimes very deep, and require continual care in order to prevent secondary infections.

One element in the clinical aspect of wounds in the cauda equina is of very frequent occurrence—that is *pain.* Persistent pain develops from the commencement, localised generally in the feet, appearing also at the level of the knee or on one side of the thigh or the leg ; stabbing pain, or a sensation as if the bone were being crushed or of torture with red-hot tongs, often eliciting moans from the sufferers, and exhausting them by insomnia.

In moderate cases these pains assume a paroxysmal form, and are affected by changes of temperature, especially by damp.

In serious cases, which are often fatal, the pain becomes an atrocious paroxysm, during which the patient, pale, gaunt, and bent, with the hands behind the neck, constantly demands analgesic cachets or injections of morphia.

As a rule wounds of this nature have a tendency to subside and to improve spontaneously, like plexus wounds of the arm.

Their variability defies any general description. But by bearing in mind the physiology of the muscles and their radicular topography, the passage of the projectile and radiographic indications, and also by making a methodical investigation of the motility of each segment of the lower limbs, and a systematic examination on several occasions of the different forms of sensibility of the whole cutaneous surface of the lower limbs, we can arrive at a precise diagnosis.

We append below a very concise *résumé* of two cases, in which we merely relate what may serve to locate the lesion :—

Case I. *Lesion of the cauda equina.*—Jean Ch——, wounded whilst lying flat by a shrapnel bullet which penetrated 3 cm. to the right of the spine of the last lumbar vertebra.

No orifice of exit. The skiagram showed a round bullet in the space between the first lumbar vertebra and the second. Immediate paralysis of both lower limbs, pain in both legs, particularly in the left foot, with retention of urine and fæces.

Six weeks afterwards the paralysis began to subside in the right leg.

Four months after the date of the wound all the movements of the right side are normal, but the power of resistance in general is diminished (the flexors of the leg, the gastrocnemius, and the extensors and the flexors of the toes are weak).

On the left side the movements of the thigh (extension on the pelvis, flexion, abduction, and adduction) take place, but are feeble ; the same applies to the extension of the leg on the thigh. All other movements of the leg and foot are abolished.

The patient cannot support himself on the left leg, and he walks with crutches.

There is pronounced atrophy of the muscles of the thighs and calves (more so on the left than on the right).

The knee- and ankle-jerks are abolished.

The cremasteric, abdominal, and anal reflexes are present. The plantar reflex is abolished.

Sensory disturbances, very marked on the left side, are found on the anterior aspect of the limb in the region of the roots L^3, L^4, L^5, S^1 ; on the posterior surface they occur in the regions L^4, L^5, S^1, S^2, S^3, S^4, S^5. They diminish in intensity towards the root

of the limb. On the right side they only consist in hypæsthesia, and occupy the region of the roots L^5, S^1 (fig. 79).

The sphincter disturbances have improved. Defæcation is normal, but the bladder still functions very badly. The retention at the

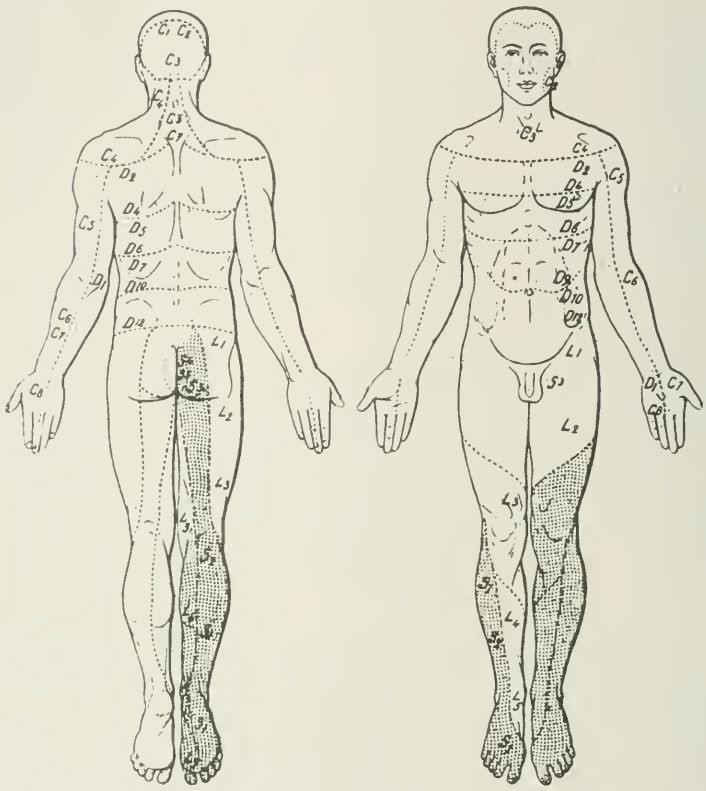


Fig. 79.—Zones of sensory disturbance in Case I. (Lesion of the cauda equina.)

onset has given way to an incontinence which is very slow in showing any improvement.

The paroxysms of pain have slightly diminished in intensity, but they occur almost as frequently as at first.

To sum up, the wound has chiefly involved the roots L^3, L^4, L^5, S^1, S^2, S^3, S^4, S^5 on the left side, and to a less extent the roots L^5, S^1 on the right.

Case II.—*Radicular paralysis of the right lumbar plexus.*—Charles R—— was wounded by a rifle bullet, which entered two fingers'

breadths above the upper border of the iliac crest, 20 cm. to the right of the vertebral line, and was not extracted. Radiography revealed a projectile 3 cm. to the right of the intervertebral disc between the fourth and fifth lumbar vertebræ, immediately below the upper border of the iliac crest.

Examination of the segmentary power of the lower limb showed the following changes :—

Dorsal extension of the foot is slightly diminished (tibialis anticus).

Flexion and extension of the knee and flexion and extension of the hip are diminished.

Abduction of the thigh is slightly and adduction greatly diminished. The gluteus maximus is fairly normal. The knee-jerk is abolished, and the ankle-jerk is normal.

The cremasteric and abdominal reflexes are normal ; the plantar reflex is flexor on the left, while on the right it is abolished.

There is slight hypæsthesia to all modes of sensation on the external surface of the thigh and on the inner side of the right leg.

The right thigh is perceptibly atrophied, and deep palpation is painful.

Electro-diagnosis shows faradic and galvanic hypoexcitability in the quadriceps, adductors, flexors of the leg, and tibialis anticus.

To sum up, it is a case of radicular paralysis of the lumbar plexus, principally involving the lumbar roots L^1, L^2, L^3, L^4 just where they unite to form the two terminal trunks—the anterior crural nerve and the obturator nerve. The paresis of the tibialis anticus and of the flexors of the leg indicate a real, though slighter, injury to the lumbo-sacral cord (L^4, L^5).

The Anterior Crural Nerve

When the projectile strikes the actual trunk of the anterior crural nerve at the highest part of the thigh, below Poupart's ligament, all the terminal branches of the nerve are affected, whence results paralysis, involving not only the quadriceps, but the other muscles supplied by the anterior crural nerve. The sensory disturbances affect a large portion of the anterior surface of the thigh and the inner surface of the leg. The knee-jerk is abolished.

Owing to the immediate dispersion of the terminal branches of the anterior crural nerve at the root of

the thigh, a projectile traversing the muscles of the anterior surface of this segment can only wound one of these branches.

Those most frequently involved are the *nerve to the quadriceps* and the *internal saphenous nerve.*

Lesions of the Nerve to the Quadriceps.—The lesion of the nerve to the quadriceps involves paralysis of this muscle, and inability to extend the leg on the thigh, and abolition of the knee-jerk. Walking is extremely difficult; the knee bends at each step, particularly going downstairs.

In these cases, notwithstanding the healing of the nervous lesion and the return of voluntary movement, lameness lasts a long time.

This persistent impotence of the knee, as Dr. Henry Meige has shown,[1] is not so much due to the weakness of the extensor muscles of the leg only as to the weakness and the distension of the aponeurotic coverings of the knee.

Attentive inspection of the affected thigh in comparison with the sound side will make these differences of structure perceptible, for "if the shape of the limb is almost normal at the upper and middle portion of the thigh, it is not the same at the lower part and all round the knee. The latter is increased in size, and is sometimes redder and colder, but the most striking feature is that one cannot distinguish either flat parts or those in relief which appear normally when the quadriceps is contracted. It is obvious that the contraction of this muscle is not accompanied by tension of the aponeurotic coverings of the knee, as is found in the healthy state. Similarly in the lower part of the thigh, one cannot distinguish during contraction the compression of the vastus internus by that thickening of the femoral aponeurosis to which Paul Richer has given the name of fascia of the vasti" (H. Meige).

Lesions of the Internal Saphenous.—The internal

[1] Henry Meige, "De certaines boiteries observés chez 'les blessés nerveux,'" *Rev. Neurolog.*, Nov.–Dec. 1915, p. 944.

saphenous, a purely sensory nerve, is more often involved than the other terminal branches of the anterior crural, although lesions of this nerve are far from common. Most frequently the projectile injures the nerve in Hunter's canal, wounding the femoral vessels at the same time. Vascular changes are then added to the nervous symptoms, and consist in purple colouration, coldness, and sometimes œdema of the foot and leg.

The lesion of the internal saphenous nerve is shown clinically by sensory, subjective, and objective disturbances.

The *subjective* disturbances consist in very sharp pain, sometimes piercing, occurring on the inner surface of the leg, the neighbourhood of the patella, the internal malleolar region, and the inner margin of the foot.

The pain, which is very intense in the earlier weeks, grows rapidly less as time goes on.

The *objective* troubles consist in more or less marked hypæsthesia to a superficial touch and to heat and cold, with occasional hyperæsthesia to pricking. They are found on the inner surface, extending over the shin to the outer surface, and on the inner margin of the foot.

The Genito-crural Nerve

Only one case of a wound to this nerve has been published by André Léri and J. Dagnan Bouveret.[1]

The lesion, due to a bullet having penetrated the lumbar region, was manifested clinically, according to these authors, by the following symptoms : sharp pain in the inguinal region at the right side of the scrotum radiating to the right thigh, retention of urine soon followed by painful micturition, and complete anæsthesia in the upper part of the right thigh and in the external iliac fossa of the same side.

[1] A. Léri and J. Dagnan-Bouveret, *Soc. de Neurol.*, May 6, 1915. *Rev. Neurolog.*, July 1915, p. 571.

The External Cutaneous, Ilio-hypogastric, and Ilio-inguinal Nerves

Lesions of the external cutaneous nerve may be shown by neuralgia of a causalgic type accompanied by smarting (Claude).

A wound involving the motor filaments of the ilio-hypogastric and ilio-inguinal nerves or their actual trunks in the lumbar region may give rise to partial paralysis of the abdominal muscles, with segmentary distension of the abdominal wall, simulating a tumour (Ch. Walther).

The Pudic Nerve

A lesion of this nerve, according to Léri, who has published an account of a case,[1] is attended by the following symptoms: painful sensations, difficulty and burning pain in micturition and defæcation, partial incontinence or at least difficulty in retaining the urine and fæces, pain in the anus, perineum, and one side of the penis, semi-erection on micturition, and flaccidity followed by contracture of the dartos.

Small Sciatic Nerve

Lesions of the small sciatic nerve cause a diminution of sensibility in the skin of the posterior surface of the thigh and calf. Sometimes, on the contrary, these lesions are accompanied by painful hyperæsthesia in the same region, as well as in the perineo-scrotal region of the small sciatic nerve (M. and Mme. Dejerine).

Table II.—Showing Motor Distribution of the Nerves of the Lumbar and Sacral Plexuses.[2]

Roots.	Nerves.	Muscles.
L^1	Nerve of the Ilio-hypogastric and ilio-inguinal	Quadratus lumborum.
		Rectus abdominis.
		External and internal oblique.
		Transversalis, pyramidalis.
	Genito-crural	Ilio-psoas.
	Anterior crural	

[1] André Léri and Édouard Roger, *Soc. de Neurol.*, July 1, 1915.

[2] Poirier and Charpy, vol. iii.

Roots.	Nerves.	Muscles.
L^2	Ilio-hypogastric and ilio-inguinal	Rectus abdominis. External and internal oblique. Transversalis, pyramidalis. Cremaster.
	Genito-crural	
	Nerve to the	Psoas.
	Anterior crural	Pectineus. Sartorius.
	Obturator	Pectineus. Adductor longus and adductor brevis. Gracilis.
	Nerve to the	(Quadratus lumborum.)
L^3	Anterior crural	Ilio-psoas. Sartorius. Quadriceps. Pectineus.
	Obturator	Obturator externus. Gracilis. The three adductors.
L^4	Anterior crural	Quadriceps (ilio-psoas).
	Obturator	Obturator externus. Gracilis. Adductor magnus and adductor brevis.
	Superior gluteal	Gluteus medius and minimus. Tensor fasciæ femoris.
	Nerve to the	Quadratus femoris.
	Great sciatic nerve	Semi-membranosus (muscles of the posterior region of the thigh).
	External popliteal	Muscles of the antero-external region of the leg, 'extensor brevis digitorum.
	Nerve to the	(Obturator internus.)
	(Inferior gluteal)	(Gluteus maximus.)
L^5	Nerves to the	Quadratus femoris. Gemelli. Obturator internus.
	Superior gluteal	Gluteus medius and minimus. Tensor fasciæ femoris.
	Inferior gluteal	Gluteus maximus.
	Great sciatic nerve	Adductor magnus. Posterior muscles of the thigh.

Roots.	Nerves.	Muscles.
L^5	External popliteal and internal popliteal .	Muscles of the leg (except the gastrocnemius and soleus). Muscles of the sole of the foot. Extensor brevis digitorum.
	(Anterior crural) . .	(Quadriceps.)
S^1	Nerves to the . .	Obturator internus. Gemelli. Quadratus femoris, pyramidalis.
	Superior gluteal . .	Gluteus medius and minimus. Tensor fasciæ femoris.
	Inferior gluteal . .	Gluteus maximus.
	Great sciatic nerve .	Posterior muscles of the thigh (adductor magnus).
	External popliteal, internal popliteal . .	Muscles of the leg and foot.
S^2	Nerves to the . .	Pyramidalis. Obturator internus.
	Inferior gluteal . .	Gluteus maximus.
	Great sciatic nerve .	Biceps, semitendinosus.
	Internal popliteal . .	Gastrocnemius and soleus. Flexor longus hallucis. External muscles of the foot.
	Internal pudic . .	Muscles of the perineum.
S^3	Superior gluteal . . .	Gluteus medius and minimus. Tensor fasciæ femoris.
	External popliteal .	Muscles of the anterior region of the leg. Peronei.
	Internal popliteal . .	Tibialis posticus. Flexor longus digitorum.
S^4	Great sciatic nerve .	Long head of biceps.
	Internal pudic . .	Muscles of the perineum.
	Nerves to the . .	Pyramidalis. Levator ani. Ischio-coccygeus.
	Internal popliteal . .	Gastrocnemius and soleus. Muscles of the sole of the foot.
S^5	Internal pudic . .	Muscles of the perineum.
	Nerves to the . .	Levator ani. Ischio-coccygeus.
S^6	Coccygeus nerve . .	Coccygeus.

CHAPTER XI

VASCULAR LESIONS ASSOCIATED WITH LESIONS OF THE PERIPHERAL NERVES

THE vasomotor, trophic, secretory, and other disturbances which we are about to describe were considered by writers before the war to be inevitable results of sections or serious lesions of the mixed nerves of the limbs.

But from the date of our earliest examinations of the wounded, with Dr Henry Meige, we observed that these symptoms appeared only in certain cases of nerve wounds, and we set about investigating their determining factor.[1]

We found that these symptoms occur only in cases where there is an arterial lesion in addition to the wound of the nerve.

In every case, therefore, in which a nervous lesion is accompanied by a reddish-purple or blue-black colour of the skin of the hand or the foot, with a glossy, tense condition of the skin, a succulent and infiltrated appearance, with considerable trophic disturbances in the nails, fingers, and toes, ending in loss of substance, injury to a great arterial trunk is to be suspected, and it is necessary to examine not only the nerves, but also the vessels of the limbs.

[1] Henry Meige and Mme. Athanassio-Benisty, "De l'importance des lésions vasculaires associées aux lésions des nerfs périphériques dans les blessures des membres," *Soc. Méd. des Hôp.*, March 12, 1915, and June 28, 1915. *Soc. de Neurol.*, November 1915.

Henry Meige and Mme. Athanassio-Benisty, "Les signes cliniques des lésions de l'appareil sympathique et de l'appareil vasculaire dans les blessures des membres," *Presse médicale*, April 6, 1916.

What means do we possess of investigating and affirming the existence of an arterial lesion? They are as follows:—

(1) *Examination of the radial pulse,* and as far as possible of the ulnar and dorsalis pedis pulses, and the pulse of the posterior tibial artery, in comparison with the sound side. In this way one can often find out the weakness or absence of the pulse on the injured side.

(2) *History of the case.*—The wounded man will nearly always tell you that he bled profusely when he was hit; that he lost consciousness; that the limb had to be tightly tied above the wound either with a dressing, a piece of string, a tie, or a tourniquet. He will tell you that in the following days he had secondary hæmorrhages necessitating more tight bandagings or strappings, which sometimes remained on a very long time; the extremity of the limb (hand or foot) became swollen, blackish, and cold. Or an artery may have had to be tied, or an arterio-venous aneurysm resected.

(3) *Investigation of the arterial pressure* by Pachon's sphygmomanometer.—There is usually a considerable fall in the maximum tension, with oscillations of weaker amplitude in comparison with the sound side. Most frequently there is a fall of several degrees in the maximum pressure, with oscillations which are at first feeble, and afterwards fairly large, and minimum pressure equal to that on the opposite side. In serious cases (destruction of the artery with defect in the collateral circulation) we find a great diminution of the maximum pressure; the oscillations are very small, hardly perceptible, and they remain so even until the armlet completely collapses, sometimes with interruptions of two or three seconds, after which the apparatus registers again. We have observed, but more rarely, with a lowered maximum pressure and normal oscillations of amplitude, a minimum pressure slightly above that of the opposite side.

(4) *Confirmation of the diagnosis by operation.*—If

in the course of an operation for a wound to a nerve you meet with a diffuse sclerotic process from which there is free hæmorrhage, making the different layers of the external integument adhere to the deeper layers, you may be certain that there is a vascular lesion, and a serious one. In fact in these cases the surgeon, when he reaches the collection of vessels and nerves, will find a complete obliteration of the arterial trunk, or else total absence of the artery, which has been destroyed at the site of the lesion, and consisting, above the wound, of nothing but a flattened tube ; in less serious cases he will meet with arterial stenosis, from compression by a fibrous band, a swollen nerve, a callus, or a ligature. In rarer cases there may be an arterio-venous aneurysm, of which the physical signs are very apparent, but which may escape notice before the operation by their localisation (in the axilla or supra-clavicular fossa).

This method confirms the diagnosis in cases where the vasomotor, trophic, and secretory disturbances are slight, and where the direct examination of the pulse and arterial pressure are negative in consequence of the re-establishment of the collateral circulation with sufficient compensation.

The disturbances arising from associated lesions of the blood-vessels and nerves may be classified under four heads :—

(1) Trophic disturbances.
(2) Vasomotor disturbances.
(3) Secretory disturbances.
(4) Sensory disturbances.

I. **Trophic Disturbances** may involve all the tissues. Proceeding from the surface to the interior :—

A.—The Skin may present various but characteristic signs of degeneration.

(*a*) Sometimes it is *fine*, *smooth*, *shiny*, and *tense*, *bluish or purple-red* in colour, recalling the "glossy skin" described by Paget. The fingers then have a

slightly succulent or œdematous appearance, especially at the tips.

(*b*) In other cases the skin is *dry*, *rough*, and *scaly*, with an ichthyotic or squamous appearance. Under these scales a purplish, dry, sometimes wrinkled and contracted skin can be perceived. The finger-tips are pointed as in sclerodermia, and the skin swells up round the nails.

(*c*) The skin may only be shrivelled, with more indurated parts forming callosities, but its colouring is always purplish-red or bluish.

Owing to the improvement of its nutrition and nerve supply the skin is liable to develop two sorts of complications—namely, *ulcerations* and *eruptions*.

Ulcerations are frequent. They occur chiefly at the end of the fingers, the inner side of the hand, and at the radial side of the index finger. In the foot they are situated at the extremity of the toes. As a result of a very slight excoriation, sometimes even without the patient being able to recall any exciting cause, blisters form which very soon burst and suppurate. When these ulcerations are seated at the ends of the fingers they encroach upon the nail, and in spite of the care taken to dress and protect them properly they take a very long time to heal, and leave behind them *retractile scars*.

In other cases, when they occur on the outer border of the index finger in the fold of joints, these ulcers have the appearance of cracks with a red floor and thick, indurated edges.

More rarely, in very serious cases, there is an actual *gangrene of the extremities*, localised to one or several fingers, encroaching upon the third phalanx, and even the second phalanx, and ending in real mutilations, as in what is known now as Raynaud's disease.

Eruptions are of more rare occurrence. They are often eczematous. We have seen a very fine example of an eruption with large scales on a finger following a wound of the superficial palmar arch.

B.—The Nails, etc.—The *nails* may present very

varied changes and deformities. They are found to be uneven, patchy, or formed of two pieces, one of which is near the base and the other near the tip, joined by an irregular tract; these two fragments have often a different thickness, substance, and colouring. Loss of one or of several nails is frequent; they are replaced by a crust, a shapeless mass of horny, thick, whitish tissue without elasticity. In other cases the nail is bluish, and invaded by the ulcerations already described.

The *hair* grows more slowly. The areas with a poor blood-supply nearly always present a less abundant pilous system than that of the healthy side.

C.—The Subcutaneous Cellular Tissue may sometimes be affected with œdema, due to a concomitant venous lesion. But what is more characteristic is the presence of a sort of *subdermic, hard, elastic infiltration*, having nothing in common with œdema, but with some resemblance to chronic trophœdema. There has to be prolonged pressure of the finger to produce even a slight pitting.

This subcutaneous thickening gives a special consistency to the extremities: the hand or the foot seem "padded."

Perhaps this infiltration is not altogether unrelated to a concomitant lesion of the lymphatic trunks which run parallel to the vessels, but we possess no means of confirming this.

D.—The Muscles.—The muscular tissue undergoes a degeneration of fibrous or fibro-sclerotic appearance; it becomes whitish, and as hard as wood. The sclerotic process passes from the superficial layers right through the muscle and down to the bone. This induration of the body of the muscle ends in the retraction of its tendon; thus degeneration of the biceps may cause retraction of its tendons and ankylosis of the elbow. This woody consistency to the touch most frequently develops in the *flexors of the fingers*, with permanent flexion of the second and third phalanges. The hand then assumes the typical

appearance observed in *Volkmann's contracture*, a deformity due, as we know, to a muscular lesion from ischæmia following compression or the prolonged enclosure of the limb in a plaster apparatus.

Besides these classical deformities we have observed in two cases a *retraction of the interosseous muscles* of the hand, shown by a permanent flexion of the first phalanx, an extension of the two others, and such a forcible adduction of the fingers that they overlapped (fig. 80). In these two cases the nerves of the forearm, the ulnar in one patient and the median and ulnar in the other, had been slightly affected. But the history in the one case (hæmorrhage and constricting band) and verification in the course of the operation in the other, demonstrated that there was total destruction of the ulnar artery, a lesion which, in our opinion, brought about ischæmic retraction of the interossei.

FIG. 80.—Retraction of the interosseous muscles following a serious lesion of the ulnar artery at the middle third of the forearm.

E.—THE ARTICULATIONS.—These do not escape the disturbance caused by vascular lesions. Ankylosis of the wrist and stiffness of the fingers develop early. Besides retraction of the tendons, which are active ligaments, the vascular defect seems to act directly on the capsule and on the soft parts of the articulations.

F.—THE BONES.—Changes also occur in the bones. We have twice seen, in cases of serious lesion of the ulnar artery, a hyperextension of the articulations of the fingers, with deformity of the last phalanges resembling chronic rheumatism.

II. **Vasomotor Disturbances.**—These are the most characteristic disturbances, and lead us to suspect that an arterial wound is present in addition to the lesion of the nerve. When they are very pronounced the skin takes on a reddish-purple tint, as if it had

been exposed to the cold for a long time, or else it is bluish-black in colour, and in the latter case it is accompanied by that succulent appearance already mentioned. The least puncture in that case, such as the pin-prick one gives in testing sensibility, makes the blood gush out.

Sometimes these vasomotor disturbances are *generalised* in the hand or foot; in other cases they are localised in one or in several fingers (very often the index finger, at other times in the three last), and they are then still more noticeable.

In other cases, particularly in those where vascular obliteration is compensated by collateral circulation, the vasomotor troubles are less pronounced, and consist only in a reddish tint of the skin of the whole extremity of the limb; but from time to time the patient passes through real attacks of asphyxia of the extremities.

The *local temperature* is always *lowered*, sometimes several degrees, in comparison with the healthy side. The hand and the fingers are *cold*, whatever the external temperature may be.

III. **Secretory Disturbances.**—These are very important to remember. In all the cases that we have met with they were evidenced by a diminution or a complete abolition of sweating throughout the region affected by vasomotor, caloric, and trophic changes.

IV. **Sensory Disturbances.**—The sensory disturbances must be divided into two classes, *subjective* and *objective*.

(*a*) The first consist in *painful phenomena*.

In serious lesions, with marked vasomotor and trophic symptoms, the element of pain does not exist, or is confined to aching and neuralgia in the ankylosed joints and in the upper insertions of the retracted muscles (coracoid process, when there is sclerosis of the biceps and internal epicondyle in the case of retraction of the flexor tendons). There is no pain either in the hand or foot. As we mentioned in considering the painful form of lesions of the median nerve, there

is a marked contrast between the *vascular hands*, which are cold, blue or purple-red, with shiny or squamous skin, and insensible both subjectively and objectively, and the "painful median" hand, which is emaciated, tremulous, hot, pink, spontaneously painful, reacting so violently to external impressions, and appearing to be in a state of perpetual combustion.

The less serious vascular disturbances (stenosis) or arterial ligatures are often accompanied by pain. This pain is sometimes of a neuralgic character, being localised near the joints or in one side of the limb; it may follow the track of a nerve, bearing a certain resemblance to the pain of obliterative arteritis, particularly that which accompanies senile gangrene. But more frequently it settles in the hand and takes on a causalgic character, with the following important differences: these painful phenomena are less intense and less lasting than in the causalgia of the median nerve. The vasomotor as well as the secretory and trophic disturbances that accompany them are of a special kind, and do not give the "vascular" hand the peculiar appearance of the "painful median" hand.

The localisation of these pains is also less definite and restricted: sometimes they affect the entire hand, sometimes its external side (the median and radial region), with a tendency to the same segmentary distribution that we shall observe in studying objective sensibility.

To explain the nature and the etiology of these pains, the hypothesis of an inflammation of the periarterial sympathetic plexus has been formed[1]—a quite probable hypothesis, and in accordance with the fact that the painful forms of vascular lesions observed by us were merely due to arterial compression or ligature.

Nevertheless, in the course of some operations (four) for painful involvement of the median nerve, with vascular disturbances following ligatures, we had

[1] M. Leriche, *loc. cit.*

occasion to find that sometimes the median only, and sometimes the median and the ulnar, had been lacerated at the time of the ligature.

If, in addition, we consider the inflammation and diffuse sclerosis caused by arterial lesions, the question arises as to whether the pain appearing in the course of vascular lesions is not due to *irritation of the periphery of the nerve*, as happens in painful lesions of the median and of the sciatic nerves. As a matter of fact, we have never encountered this pain without an associated lesion of the median or the sciatic nerve.

(*b*) The disturbance of *objective sensibility* consists in complete, extensive anæsthesia of segmentary type, with constantly changing localisation, and bearing no relation to the peripheral distribution of the nerve filaments. This anæsthesia sometimes occupies the extremity of all the fingers, sometimes the whole of the three last fingers or all the index finger, or the entire hand or foot. It affects the regions where the vasomotor, caloric, and trophic disturbances are very marked.

The arteries most often wounded in the cases observed by us were, in order of frequency :—

1. The brachial artery in the arm, with an associated lesion of the median nerve alone, or of the median and ulnar nerves.

2. The axillary artery, with a complex lesion of the brachial plexus, or an isolated wound of the median and ulnar nerves.

3. The radial artery, with a wound of the median nerve in the forearm, or of the median and sensory branch of the musculo-spiral.

4. The ulnar artery, with a lesion of the ulnar nerve alone (retraction of the interossei), or of both the median and ulnar.

5. The popliteal artery, with lesion of the external and internal popliteal nerves (gangrene of the toes).

6. The femoral artery, with a wound of the great sciatic nerve in the lower part of the thigh (gangrene of the last three toes).

In conclusion, we may mention an interesting therapeutical point :—

Surgical interventions on limbs presenting serious vascular disturbances, with the object of liberating or suturing the wounded nerves, have nearly always a favourable effect on vasomotor, trophic, and caloric symptoms. The surgeon is obliged to cut out what remains of the nerve-trunks from very indurated and vascularised fibrous tissue ; although he is obliged at the same time to sever many very much dilated small tributary arteries, the intervention has never, in our experience, been followed by any aggravation whatever. On the contrary, the wounded notice an improvement in the vascular symptoms affecting the extremities in the first weeks following the operation. In fact, a real improvement follows the release of the nerves from the adhesions. The coldness diminishes as well as the trophic changes and the vasomotor phenomena.

CHAPTER XII

CRANIAL NERVES

Cases in which wounds of the face and of the upper and lateral part of the neck are accompanied by lesions of one or several cranial nerves are not uncommon.[1]

Numerous observations of this kind have been published by different authors, notably by André Thomas,[2] Morestin,[3] Collet,[4] Vernet,[5] etc.

We shall say a few words about the changes that must be investigated to arrive at a diagnosis of lesions of the nerve-trunks most often affected.

The complicated and capricious course taken by projectiles makes a general description of the lesions of the cranial nerves an utter impossibility.

The Trigeminal Nerve

Lesions of this nerve, which is at once motor and sensory, are not uncommon. We may remind the reader that it supplies a motor branch to the *masticatory muscles*, and from the sensory aspect supplies the skin of the face comprised between the region of the great occipital nerve behind and the branches of

[1] In this necessarily summary account we do not include lesions of the optic nerve, the motor nerves of the eye, nor of the auditory nerve.

[2] André Thomas, "Multiple Paralyses of the Cranial Nerves," *Soc. de Neurol.*, July 1, 1915.

[3] H. Morestin, *Soc. de Chir. de Paris*, June 23, 1915, June 30, 1915, January 5, 1916.

[4] Collet, *Lyon Médical*, April 1915.

[5] Maurice Vernet (of Lyons), *Soc. Méd. des Hôp.*, February 18, 1916.

the superficial cervical plexus in front and laterally (fig. 62).

In addition, it gives sensibility to the gums, palate, inside of the cheeks, nasal cavities, eyeball, lips, teeth, and floor of the mouth.

By its lingual branch, which anastomoses with the chorda tympani (a branch of the facial), it gives sensibility to the anterior half of the dorsal surface of the tongue.

It is therefore necessary to examine all these regions attentively, in case the projectile has traversed the regions containing branches of the trigeminal.

When the *inferior maxillary nerve* may have been injured it is necessary to examine the masticatory muscles. The patient must be made to clench and unclench the teeth, and while he is doing so the masseters and the temporals must be palpated ; then he must execute the movements of the lower jaw (elevation, propulsion, retropulsion, and movements of latero-flexion). In cases of paralysis of all the masticatory muscles of one side, movement of the whole jaw towards the healthy side is observed on inspection.

Paralysis of the masticatory muscles is not very frequent. It is generally due to the injury of one or several offshoots of the sensory terminal branches of the trigeminal nerve, shown by a patch of anæsthesia or hypæsthesia. Sometimes there may be painful hyperæsthesia.

In some cases a patch of alopecia has been found in one of these hyperæsthetic zones following a lesion of the trigeminal nerve and of the auricular and mastoid branches of the superficial cervical plexus. Paralysis and irritation of the inferior dental nerve may have played a part in the pathogeny of this symptom by reflex action (André Thomas).

André Thomas also regards a lesion of the lingual nerve or of the chorda tympani as responsible for vasomotor disturbances in the half of the tongue corresponding to the injured side. These vasomotor

phenomena consist in a pallor of the lingual mucosa, and a poorer blood-supply to the lower surface of the tongue, where the ranine vein appears smaller than that of the healthy side.

Besides the disturbances of general sensibility, *gustatory sensibility* may also be affected in the course of a lesion of the trigeminal nerve. Search should therefore be made for this on the anterior segment of the dorsal surface of the tongue.

In connection with lesions of the trigeminal nerve, we must examine a "sympathetic" syndrome which we had occasion to observe, with Dr Monbrun, in patients who had undergone amputation of the anterior segment of the eye in consequence of wounds to that organ.[1] Several weeks or several months after a wound to the eye has been operated on and perfectly healed, it is not unusual to meet with a number of disturbances which are nearly always identical in the different cases, and vary only in their intensity.

They consist of pain in the head, localised in the vertex or nape of the neck, starting from the root of the nose, or sometimes from the region of the orbit.

These pains are more or less continuous, but they also occur in paroxysms, in the course of which vasomotor disturbances appear, characterised by redness of half or the whole of the face and the neck, attacks of flushing, a feeling of anguish, and slight dizziness.

These pains, which the sufferers compare to blows of a hammer, rending of the flesh, and burning, are increased or provoked by the same circumstances as causalgia in wounds of the median or sciatic. They are intensified by noise, light, and emotion, and most of all by *atmospheric heat*.

Secretory disturbances are sometimes seen in the course of the paroxysms, and consist of hypersecretion of sweat on the face.

It is curious to note the existence of hyperalgesic cutaneous zones, sometimes at the nape of the neck,

[1] M. Monbrun and Mme. Athanassio-Benisty, *Soc. de Neurol.*, May 4, 1916. *Rev. Neurolog.*, June 1916, p. 906.

but more often at the vertex, corresponding, according to Head's diagrams, to lesions of the posterior segment of the eye.

In these zones the wounded feel either itching, burning, or persistent and very disagreeable tingling.

The intensity of these painful symptoms always reacts on the psychical condition of the patient, who becomes uneasy, irritable, solely preoccupied by his sufferings, exasperated sometimes even to the thought of suicide. Struck by the resemblance of these phenomena to those which Dr Henry Meige and ourselves have described in lesions of the median and sciatic nerves, we have endeavoured to discover their starting-point.

Dr Monbrun discovered in all these cases the existence of a stump in which the ophthalmic ganglion and its afferent and efferent branches might be imprisoned and irritated.

The *irritation of the cephalic sympathetic* at this point might, we think, explain the appearance of these painful, vasomotor, and secretory symptoms, but the removal of the stump did not bring about, as we hoped, any considerable improvement in these symptoms.

But it would be desirable that total enucleation of the wounded eye should be performed at once in every case of serious ocular wound before awaiting the outbreak of the intense and obstinate disturbances of which we have spoken.

The Facial Nerve

The facial nerve is fairly frequently injured in wounds of the parotid region and of the cheek. Moreover, wounds of the cranium, particularly in the temporo-frontal region, are very often accompanied by paralysis of the motor filaments to the orbicularis palpebrarum or frontalis.

The division of the nerve into two branches, the *temporo-facial* and the *cervico-facial*, and the numerous

ramifications they send out to the superficial muscles of the face, offers a ready explanation of wounds being localised in the inferior or superior facial nerve, and even in some of their terminal fibres.

But it may happen that a lesion of the actual

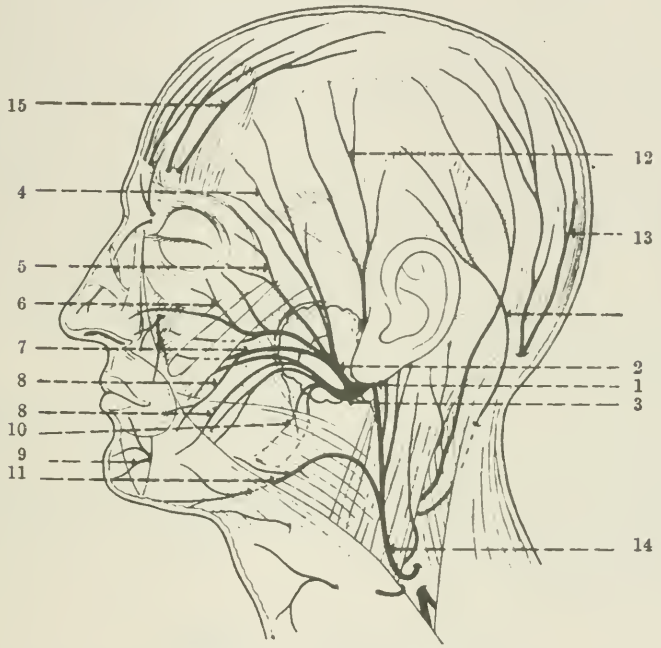


FIG. 81.—1. Facial nerve. 2. Temporo-facial division. 3. Cervico-facial division. 4. Frontal branches. 5. Palpebral. 6. Sub-orbital. 7. Superior buccal. 8. Inferior buccal. 9. Mental. 10. Cervical. 11. Transverse cervical. 12. Auriculo-temporal. 13. Sub-occipital nerve. 14. Auricular. 15. Supra-orbital.

trunk of the facial nerve gives rise to an incomplete dissociated paralysis of the nerve, which is localised by preference in the fibres of the inferior facial.[1]

Dr Moure explains this fact by the following hypothesis: the trunk of the facial nerve is composed of two kinds of fibres, the superficial ones supplying the muscles of the lower part of the face, and the others

[1] E. J. Moure, "Paralysies faciales de la guerre," *Presse médicale*, April 13, 1916.

occupying the centre and destined for the upper muscular group.

The first, which are the more sensitive, form a covering for the second, and are therefore more exposed to external irritation.

The diagnosis of total or partial paralysis of the facial nerve is easy. The expressionless set appearance of half of the face, the flabbiness of the cheek, the effacement of wrinkles on the forehead and of the naso-labial fold, the deviation of the mouth, drooping of the lower lid, the lowering of the eyebrow, inability to close the eye on the paralysed side if the superior facial has been also affected, and the turning of the eyeball upwards and outwards when trying to close the eye, are some important signs. The asymmetry of the face is exaggerated during movement, when the patient speaks, laughs, or makes grimaces.

Electrical examination, repeated at fairly long intervals, will enable one to ascertain the intensity of the lesion, and under these conditions one may decide upon the advisability of surgical intervention directly on the nerve itself (Moure), or a palliative operation on the soft parts (fixation to resisting structures (Morestin)).

Other Cranial Nerves

The glossopharyngeal is a mixed nerve giving motor fibres to the pharynx and soft palate, and sensory fibres to the pharynx and tongue.

When it is wounded one or both of the following symptoms may be observed : *paralysis of the constrictor of the pharynx* and *alteration of taste* (Collet, Maurice Vernet).

Paralysis of the constrictor of the pharynx is shown by difficulty in deglutition of solids in the lower part of the pharynx, so that the patient is obliged to facilitate this deglutition by the ingestion of fluid.

At the same time the *soft palate* may be involved.

In case of paralysis half the soft palate is immobilised and deviated to the sound side, with nasal regurgitation of fluids and a nasal voice. In addition to these signs we notice that the *posterior wall of the pharynx* on the affected side shows a *movement towards the healthy side* (Maurice Vernet).

Examination of taste by comparison with the sound side shows, on the posterior part of the tongue, a diminution or abolition of the perception of bitter substances (quinine), and a diminution or retardation as regards salt or sugar.

Reflex sensibility (nausea reflex, reflex of the soft palate) and sensibility to touch of the soft palate and the posterior wall of the pharynx may be diminished or abolished on the affected side.

The Pneumogastric Nerve.—A lesion of this nerve may be accompanied by various disturbances. The most usual and the most characteristic are *laryngeal* and *cardiac disturbances.*

The first are shown by alterations in the voice, which is weaker, bitonal, and sometimes wheezing.

On laryngoscopic examination the vocal chord on the wounded side is found in the intermediate or cadaveric position. Often the vocal chord of the healthy side, by an effort at compensation, goes beyond the median line in front of the paralysed vocal chord.

The cardiac disturbance is shown by an acceleration of the pulse. O. Josué and Jean Heitz[1] regard irritation of the pneumogastric nerve as the cause of attacks of extra-systoles occurring after rapid or fatiguing exercise in a patient under their observation who had been wounded in the neck by a shrapnel bullet.

Attacks of convulsive coughing, which appears to be due to pharyngo-laryngeal hyperæsthesia from irritation of the nerve, may also be met with.

Lastly, another sign of a lesion of the pneumo-

[1] *Archives des maladies du cœur, des vaisseaux et du sang*, September 1915.

gastric nerve, according to Vernet, is pain on pressure on the corresponding ala of the thyroid cartilage.

The Spinal Accessory Nerve.—This exclusively motor nerve divides, soon after it issues from the cranium, into two terminal branches, one internal, which immediately joins the pneumogastric nerve, thus forming the motor root of the latter; the other, external, which is intended for the innervation of the trapezius and sterno-cleido-mastoid.

The internal branch supplies by itself the muscles of the larynx and those of the soft palate, the vagus nerve being purely sensory. According to some authors, this internal branch is the origin of the cardiac nerves of the vagus. The laryngeal and cardiac disturbances previously described would therefore be due to the lesion of the fibres of the spinal accessory contained in the interior of the vagus nerve.

Lesion of the fibres of the external branch entails a paralysis of both trapezii and sterno-mastoids, the signs of which we described in studying the lesions of the cervical plexus.

The hypoglossal is a purely motor nerve; it supplies the muscles of the tongue, the genio-hyoid muscle and the sub-hyoid muscles. It is rather frequently injured in the supra-hyoid region, and more rarely in the latero-pharyngeal space.

Its lesion is shown by a *hemiparesis of the tongue* The corresponding half of the organ is reduced, shrivelled, and its tip is deviated towards the paralysed side.

This deviation is increased when the patient tries to put the tongue out. The mucous membrane is thrown into large folds. On palpation the paralysed half seems soft and flabby; electrical examination shows a reaction of degeneration. When the two hypoglossal nerves have been wounded by a projectile traversing the supra-hyoid region from one side to the other the tongue is completely paralysed. It is flaccid and immobile; the patient cannot put out the tip, nor draw it back, nor raise it.

The wounded man swallows his saliva with great difficulty, dribbles constantly, and is obliged to have a handkerchief at hand to keep his face dry. He speaks with difficulty in a thick, scanning voice, and has great difficulty in feeding himself.

When the hypoglossal nerve is paralysed at the base of the cranium, we also observe an appreciable paralysis of the sterno-hyoid muscle, with electrical disturbances.

Professor Collet (of Lyons) gives the name of *complete hemiplegic syndrome* of the last four nerves to a combination of symptoms due to a complete lesion of the ninth, tenth, eleventh, and twelfth cranial nerves, such as may be caused by a wound in the uppermost part of the latero-pharyngeal space. This syndrome is shown by the following signs: lingual hemiplegia, laryngeal hemiplegia, unilateral paralysis of the trapezius, hemiplegia of the soft palate and pharynx, with disturbance of taste, cardiac rhythm, and some ocular symptoms, probably due to involvement of the cervical sympathetic nerve.

This syndrome would thus constitute a new type of *associated laryngeal hemiplegia* besides the pure laryngeal hemiplegia, the syndrome of Avellis (larynx and soft palate), Schmidt's syndrome (larynx, soft palate, trapezius), Jackson's syndrome (larynx, soft palate, and tongue), and the syndrome of Tapia (larynx, tongue).

CERVICAL SYMPATHETIC

Although often associated with wounds of the brachial plexus, lesions of the cervical sympathetic from wounds in war may also be observed separately.[1]

These lesions react on the organ of vision in quite a peculiar manner. The ocular symptoms are as follows: *more or less considerable myosis on the affected side, sinking in of the eyeball, and diminution of the palpebral fissure.*

[1] Couteaud, *Soc. de Chir.*, February 9, 1915.

The visual function does not appear to be influenced by the sympathetic lesion. In the four cases described by Couteaud, visual acuity was normal, there was no disturbance of accommodation, and no modification of the membranes or of the media.

Besides the ocular phenomena, one may observe a vaso-dilatation of the cheek on the affected side, which is hotter and redder than the other. More rarely one may find a diminution of salivation.

SECTION II

TREATMENT & REPAIR OF NERVE LESIONS

Treatment and Repair of Nerve Lesions

CHAPTER I

MACROSCOPICAL AND MICROSCOPICAL LESIONS OF INJURED NERVES

BEFORE considering the naked-eye and microscopical changes which nerves undergo as a result of war wounds, a brief description must be given of the current views as to the structure of nerves, the mode of their degeneration when subjected to various forms of traumatism (crushing, compression, and section), and the course of their cicatrisation and repair. The following description is based on the interesting researches of Nageotte[1] and the article by Prof. Pitres.[2]

A nerve consists of nerve fibres, which constitute the active element of this organ, and of connective tissue. Within the nerve these fibres become grouped in bundles. Each bundle is enclosed in a connective-tissue sheath, the *lamellar sheath*; the nerve-trunk itself is formed by the aggregation of these bundles, and is encased in a sheath consisting of connective tissue called the *neurilemma*.

[1] J. Nageotte, *Comptes Rendus de la Société de Biologie*, vol. lxxxiii., and *Revue Neurologique*, July 1915.

[2] A. Pitres, "Sur les processus histologiques qui président à la cicatrisation et à la restauration fonctionnelle des nerfs traumatisés," *Journal de Médecine de Bordeaux*, December 1915.

The nerve fibre of the cerebro-spinal nerves is formed by three distinct elements : the *axis-cylinder*, the *medullary sheath*, and the *sheath of Schwann*.

The *axis-cylinder* or *axone* is a ribbon-like, glistening tract made up of contiguous fibrillæ, which give it the appearance of longitudinal striation. Silver nitrate causes a black deposit on its surface.

The *medullary sheath* encloses the axis-cylinder, forming a sort of sleeve, interrupted at intervals corresponding to the *annular constrictions*. Myelin reduces osmic acid, and takes on a black colour. Finally, outside the medullary sheath is the *sheath of Schwann*, which is a delicate, transparent membrane bearing on its inner surface nuclei surrounded by a thin layer of protoplasm.

At the level of each annular constriction the nerve fibre consists solely of the axis-cylinder and the sheath of Schwann.

The blood-vessels and lymphatics of the nerve circulate between the lamellar sheaths.

Section of a Nerve in the Animal

In the first instance we shall consider the case of a nerve divided under aseptic precautions and whose proximal and distal ends have remained in close proximity to each other. Immediately after division there is an effusion of blood containing débris from the surrounding tissues.

Provided suppuration does not occur, in a few days a more or less exuberant mass of fibro-cicatricial tissue is formed, the size of which depends upon the distance between the two severed ends of the nerve.[1]

During this time the nerve tissue proper of both the central and peripheral ends undergoes changes.

All the nerve fibres situated below the point of section—*i.e.* the whole of the lower segment of the nerve—undergo a series of histological modifications, known under the term of *Wallerian degeneration*,

[1] A. Pitres, *loc. cit.*

from the name of the physiologist Waller, who described this condition.

Before the end of the first day the axis-cylinders of the nerve fibres begin to swell. At first this swelling is limited to the immediate neighbourhood of the wound, but subsequently it gradually travels down to the terminal filaments of the nerve. At the same time the fibrils constituting the axis-cylinder undergo changes, and grow sinuous and varicose.

From the second day onwards the nuclei of the sheath of Schwann tend to hypertrophy, their protoplasm increases in size and attacks the myelin, which becomes broken up into irregular, brilliant globular masses arranged around the axone.

Towards the fourth day the nuclei of the interannular segments start on a phase of active proliferation, while the myelin becomes more and more broken up, until, by about the fifteenth day, it disappears completely.

Wallerian degeneration ends with the loss of excitability of the nerve fibres, which occurs three or four days after division of the nerve. This loss of nervous excitability seems to affect all methods of stimulation (electrical, chemical, and mechanical). The muscles do not become irresponsive to stimuli until much later, several weeks or even months after the nerves have grown inexcitable (Pitres).

Synchronously with the development of this process of degeneration a *process of regeneration* starts.

The lower segment, by undergoing the changes described above, prepares to receive the newly formed axis-cylinders about to be sent out from the end of the upper segment.

This process of regeneration progresses very slowly, and is only complete when the motor function of the nerve has been restored. We shall therefore have to describe this transformation in several phases.

From the first few hours after division of the nerve a number of slender fibrillæ—the future axis-cylinders in the central extremity of the nerve—are seen to

separate themselves from the lateral walls of the injured axis-cylinders immediately above the level of section. This is the initial phase of regeneration, and is termed *Perroncito's phenomenon.*

These fibrillæ grow actively, and penetrate the fibro-cicatricial tissue which intervenes between the cut ends of the nerve. They are attracted by a sort of positive chemiotaxis towards the inferior extremity of the divided nerve. They increase in length and

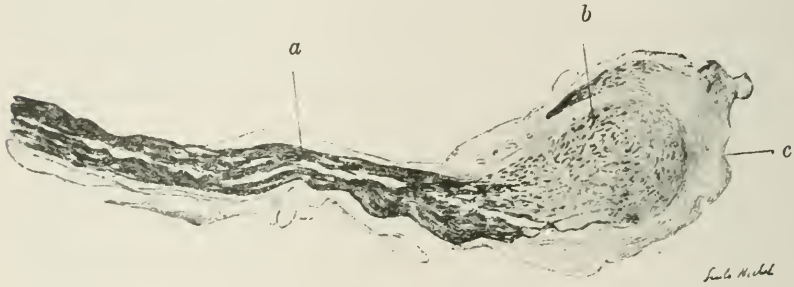


Fig. 1.—Experimental neuroma in a rabbit (upper end).

Section and attrition. Cap of loose tissue on the end of the nerve; this cap has not been colonised by the fibres. *a*, afferent end; *b*, neuromatous part; *c*, cap of loose tissue. (After Pierre Marie and Foix.)

thickness, and their terminal extremity has a club-shaped swelling.

It should be noted that they do not penetrate the fibro-cicatricial tissue directly, but by spreading out in the form of an interlacing network (fig. 1).

It will be readily understood that when the intervening mass of fibro-cicatricial tissue is very exuberant and rich in fibres (such as occurs as a sequel to inflammations and protracted suppuration) the axis-cylinders of the central segment have great difficulty in getting through to reach the peripheral segment.

On the other hand, when the inflammation has been slight, the two cut ends being separated only by a short interval, cicatrisation by first intention may be perfect, and after regeneration the nerve may appear

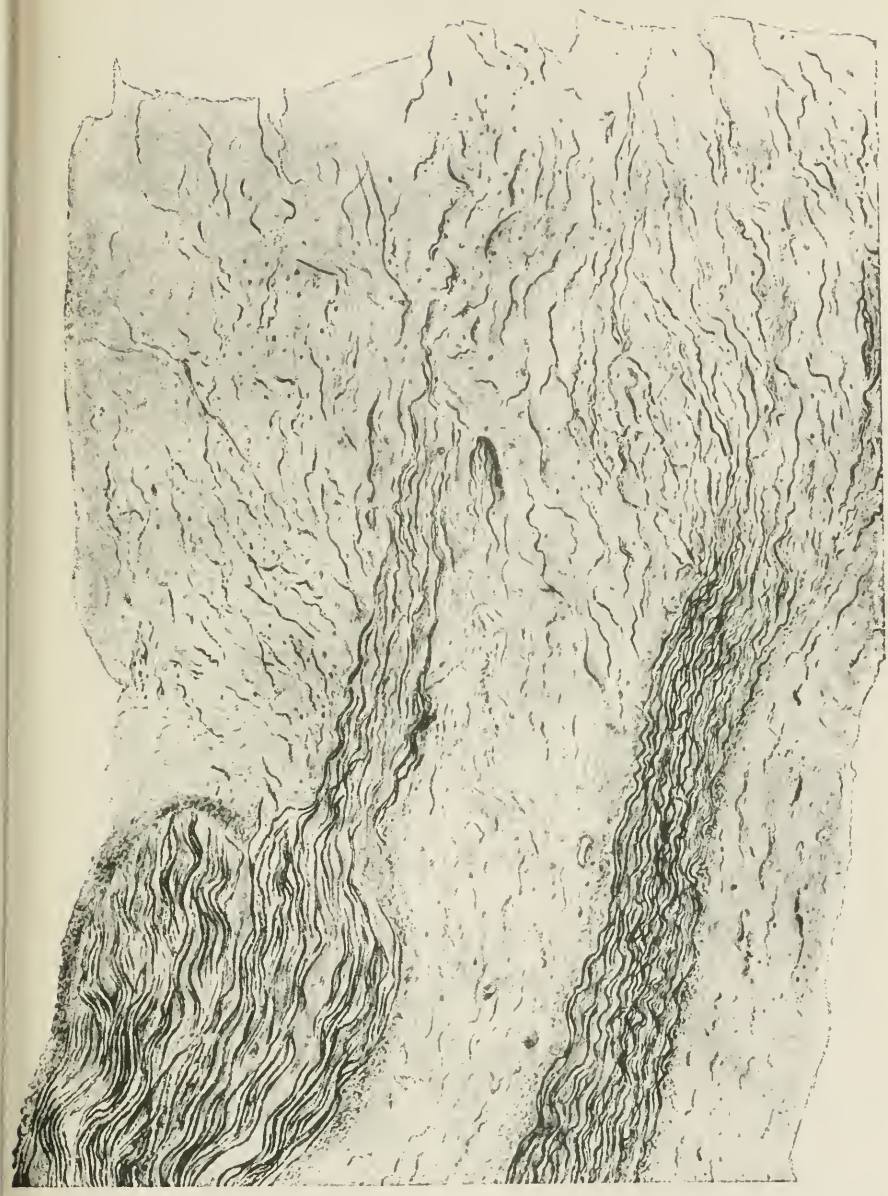


PLATE I

Excessively sclerotic pseudo-neuroma. (After Pierre Marie and Foix.)

Rupture of the lamellar sheath. Spreading and dislocation of the fibres in the part of the nerve that has suffered most injury. Their structure is relatively preserved as well as their direction towards the lower end.

hardly thicker or more vascular at the site of the injury than elsewhere.

If the two segments are separated by more than 15 or 20 millimetres, a fibro-cicatricial bridge, or tract adherent to the neighbouring tissues, is formed connecting the two swollen ends of the nerve with each other.

The swelling of the central end, which is always the larger, represents a *neuroma*, formed by the proliferation of the sheaths of Schwann and the young axones or *neurites* (Nageotte).

The peripheral segment, which shows a smaller swelling, is, on the other hand, made up solely of the exuberant proliferation of the nuclei of the sheath of Schwann, which terminates in a network of sheaths into which the young neurites penetrate.

Nageotte calls this swelling a *glioma*, since it is formed solely from the sheath of Schwann, which constitutes the neuroglia of the nerve.

This glioma grows rapidly, and presses straight on towards the central end.

In short, the regeneration of a divided nerve is due on the one hand to the proliferation of the axis-cylinders of the central end, and, on the other hand, to the proliferation of the nuclei and the protoplasm of the sheath of Schwann, which ends in the formation of new sheaths ready to receive the young neurites (axis-cylinder processes) both at the central end and in the whole of the peripheral segment.

"*The neuroglia* (*sheath of Schwann*) *builds up the nerve, and the axis-cylinders proceed to lodge there*" (Nageotte).

What happens when the wound has caused considerable destruction of the nerve and the two ends are completely separated from each other ?

The central bud divides and spreads out, the young axis-cylinders are folded back and rolled up, grow out at the sides or even turn round and grow in the opposite direction (fig. 2).

In the peripheral segment the sheaths remain

empty, and wither sooner or later. Occasionally, however, despite the intervention of dense fibro-cicatricial tissue, a few axis-cylinders reach the inferior segment by devious paths and neurotise, *i.e.* regenerate it by penetrating into the prepared sheaths awaiting them there. This phenomenon was discovered by Ranvier.

As soon as these neuro-fibrillæ reach the sheaths they grow rapidly, become coated with myelin, and gradually progress up to the terminal motor or sensory extremities of the nerves.

It is only then that any restoration of function

Fig. 2.—Section and attrition in the rabbit.

a, cap of connective tissue and muscle; *b*, nerve in course of degeneration. The cap of connective tissue and muscle is mainly formed by the proliferation of Schwann's sheaths, but inflammatory scleroses and muscular adhesions play an important part in the injured nerves. (After Pierre Marie and Foix.)

appears in the nerve. The phase that requires most time is therefore the journey through the mass of fibro-cicatricial tissue, which presents the most formidable obstacle to the regeneration of the lower end in the first place, and later to the functional recovery of the whole nerve.

The experiments of Van Lair have shown that a nerve divided under aseptic precautions in an animal, both cut ends remaining in contact, unites by first intention, and recovers its power to conduct impulses at the end of eight to eleven months. If the space between the cut ends is about 1 centimetre repair takes longer, and is not complete until about the end of fourteen months, but it may be delayed up to thirty months if the space between the cut ends is 2 centi-

metres. No recovery takes place when the interval is 4 centimetres (Pitres).

From Nageotte's researches[1] it appears that in addition the following practical conclusions can be deduced; although logically justified *a priori*, an operation is harmful which attempts to assist the junction of the two ends of the nerve and to canalise the growth of neuroglia on the end of the lower segment, for instance, by ingrafting a vein. In such cases the lower sprout is more delicate, and a large number of axones of the upper end do not reach it, but spread out in all directions in spite of the canalisation.

Nageotte also considers that a tight suture is less favourable than a loose one, allowing the growth of a fibro-cicatricial mass which will be well honeycombed by inter-anastomosing fibres of regeneration, thus ensuring a more perfect neurotisation of the inferior segment.

The above is a short *résumé* of the facts known about the degeneration and regeneration of nerves in animals after experimental division or crushing.

Injuries of Nerves by War Wounds

How do nerve injuries progress which are due to wounds received in war, and what is the naked eye and microscopical appearance of the injured nerve?

Our knowledge of this remains incomplete in spite of elaborate investigations, since it is difficult to control all the various phases of this process.

On the other hand, it may be said that every time a nerve is injured by a war traumatism the reaction is peculiar to that case, since the conditions of the wound and the inflammation of the surrounding tissues are different on each occasion. In spite of a superficial resemblance in appearance and structure the cicatrix

[1] J. Nageotte, "Le processus de la cicatrisation des nerfs," *Revue Neurologique*, July 1915.

of a nerve which has been sutured, resected, or freed does not always take the same clinical course.

Nevertheless, two years of observation and research have established certain facts of an anatomical and pathological nature. We propose to describe them by giving a *résumé* of the work of Pierre Marie and Foix,[1] Claude, Vigouroux and R. Dumas,[2] Mme. Dejerine and J. Mouzon,[3] and Jourdan and Sicard.[4]

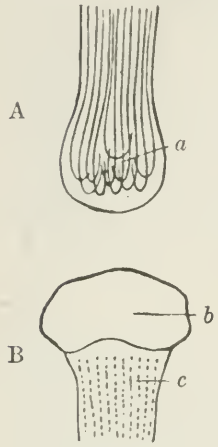


Fig. 3.
Complete section.

A, Upper neuroma; *a*, turning back of the fibres. B, Lower neuroma; *c*, region of the degenerated fibres; *b*, cap of connective tissue and muscle. (After Pierre Marie and Foix.)

Following Pierre Marie and Foix, we shall distinguish three main types of lesions :—

1. Complete section.
2. Pseudo-neuroma of attrition.
3. Lateral notch.

Among these three main forms of lesions, the *pseudo-neuroma of attrition* is the one most frequently seen. The lateral notch occurs fairly often. Complete section, on the contrary, is relatively rare, and only occurs, according to Claude, Vigouroux and R. Dumas, in about 22 out of every 100 cases. We shall then proceed to describe four types of accessory lesions :—

4. Complete section with pseudo-continuity.

[1] Pierre Marie and Foix, "Indications opératoires fournies par l'examen histologique des nerfs lésés par plaies de guerre," *Presse médicale*, Jan. 31, 1916.

[2] Claude, Vigouroux and R. Dumas, "Étude anatomique de cent cas de lésions traumatiques des nerfs des membres," *Presse médicale*, March 4, 1915.

[3] M. and Mme. Dejerine and M. J. Mouzon, "Les lésions des gros troncs nerveux par projectiles de guerre. Les différents syndrômes cliniques et les indications opératoires," *Presse médicale*, May 10, July 8, August 30, 1915.

[4] Jourdan and Sicard, "Étude macroscopique et microscopique des lésions des nerfs par blessure de guerre," *Presse médicale*, July 29, 1915.

PLATE II

Pseudo-neuroma of attrition. (After Pierre Marie and Foix.)

At the lower part of the pseudo-neuroma there is a partial absence of myelin, but the arrangement of the fibres is preserved.

5. Lateral pseudo-neuroma with or without a lateral notch.

6. Partial neuroma (intra- or juxta-nervous).

7. Simple nerve induration.

1. **Complete Section.**—As soon as the surgeon finds the nerve-trunk in the operation wound either above or below the probable seat of the lesion, according to the course of the projectile, he will see by following it up that in the majority of cases where complete section has occurred it plunges "suddenly and at an angle" into the wound (Pierre Marie and Foix). An appearance such as this is strongly in favour of complete nerve division. In fact, by following and freeing the nerve it will be found to be severed, with both ends separated, swollen, and more or less adherent to the surrounding fibro-sclerotic tissue (fig. 3).

Histologically, the upper swelling, or "neuroma," when the injury has occurred sometime previously, is composed of adult medullary sheaths possessing relatively normal axis-cylinders (P. Marie and Foix).

Fig. 4.

Complete section. Old-standing upper neuroma. (Magnification, 7·5 diameters.)

The neuroma is wholly composed of myelinated fibres. In its uppermost part (*a*) the fibres are parallel, in the middle part (*b*) they spread and diverge, in the lower part (*c*) they run in all directions. This part is, therefore, not suitable for suture. (After Pierre Marie and Foix.)

This neuroma is encapsuled and surrounded by a coat of connective tissue, against which the young nerve fibres impinge and are obliged to retrace their steps (fig. 4).

Thus the regenerated nerve fibres follow a longitudinal and parallel course in the upper part of the neuroma, and then diverge and scatter in all directions, a large number of them taking an upward course.

FIG. 5.
Pseudo-neuroma of attrition.
a, region of healthy fibres; *b*, region of degenerated fibres. (After Pierre Marie and Foix.)

Pierre Marie and Charles Foix have been able to reproduce this phenomenon in animals by dividing a nerve which had been previously compressed by forceps, contusion being a necessary factor to produce the inflammatory condition which is always very intense in war wounds.

The lower peripheral extremity of the cut nerve is also enlarged, but usually less so than the upper segment. This lower pseudo-neuroma, or "glioma," to use the term employed by Nageotte, consists in war wounds of a shell of fibro-connective tissue, very frequently containing muscular fibres, and covering a nerve in complete Wallerian degeneration.

Pierre Marie and Foix, as well as M. and Mme. Dejerine and J. Mouzon, have drawn attention to the phenomena of ascending degeneration of the myelin sheaths and of the axis-cylinders of the central end. This fact should be remembered, as it may help to explain some cases of complete division, in which nerve suture, carried out under the most promising conditions, followed by complete healing of the wound, nevertheless ended only in failure.

2. **Pseudo-neuroma of Attrition.** — This lesion, which occurs in the majority of nerve wounds caused by projectiles, has the naked-eye appearance of a

nodule or cicatricial swelling, more or less olive-shaped and of regular outline, situated in the course of a nerve which has maintained its continuity (fig. 5).

FIG. 6.—Pseudo-neuroma of attrition. (Magnification, 7·5 diameters.)

The increase in size is due to the over-production of interstitial tissue. Myelinated strands which have kept their direction are still easily recognisable. *a*, region grazed by the bullet; *b*, myelinated fibres in definitely grouped fasciculi; *c*, small blood-vessel affected by endarteritis. (After Pierre Marie and Foix.)

This is what Sicard and Jourdan call the "*trans-lesional nodule*," Claude, Vigouroux and Dumas "*incomplete section*," M. and Mme. Dejerine and J. Mouzon "*cheloid of the axis-cylinder*." These last observers consider that this lesion is equivalent to

total section when the clinical signs are severe, since their histological investigations have demonstrated to them that this "cheloid" was of such a fibrous and tough nature, that it had not yet been pierced by the axis-cylinders of the central end 113 days after the original injury, and that the peripheral end was in complete Wallerian degeneration. Resection followed by suture, according to them, is the only logical operative treatment suitable in such cases.

We know, however, nowadays that this kind of anatomical lesion is found in the great majority of

FIG. 7.—Experimental pseudo-neuroma of attrition in the rabbit.

a, afferent end ; *b*, swollen pseudo-neuromatous end ; *c*, efferent end. (After Pierre Marie and Foix.)

nerve injuries, that nerve recovery is an exceedingly slow process when the injury has attained a certain degree of severity, as two years of observation have proved, and that, finally, the vast majority of nerve injuries recover without surgical interference, or after simply freeing the nerve. For all these reasons we incline to the opinion of Pierre Marie and Foix, whose histological arguments agree in all points with clinical observations and the results of operative interference to show that the existence of a pseudo-neuroma in the course of a nerve is not equivalent to a complete section of the nerve.

"Histologically, the pseudo-neuroma is mainly composed of fibrous tissue separating the bundles of nerve fibres, and thus causing the increase in size (fig. 6).

"There is no multiplication of nervous tissue, and this formation does not deserve the name of neuroma. It is a kind of small fibroma, which readily heals.

"With regard to the lesions, three zones can be distinguished: an upper zone, in which the fibres descend by spreading, separated by the proliferation of connective tissue; an intermediate zone, where the lesion is at its maximum, and from which point onwards the fibres degenerate; and a lower zone, where the fibres exhibit more or less complete secondary degeneration.

"When the lesion is at its height two things may happen: either the fasciculi persist, the medullary fibres growing scarcer or disappearing completely; or the lamellar sheath is torn, and the sheaths of Schwann are then found freed, spread out, and partly dislocated.

"Regeneration appears to us to be very probable in those cases where the nerve fasciculi traverse the injured part, even when they are completely degenerated, without being spread out.

"When spreading out takes place the prognosis, although less satisfactory, still remains relatively favourable. Considered from the point of view of the number of sheaths preserved intact the nerve is amply sufficient, for beside the full medullated sheaths, there are empty sheaths whose structure can easily be made out, and which will regenerate in their turn.

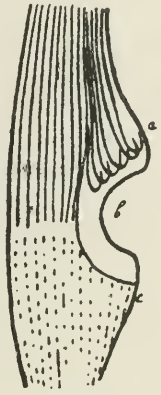


FIG. 8.
Lateral notch.

a, upper lip of the notch with the fibres turning back; *b*, notch; *c*, lower lip of notch. The region shown in white consists of sclerotic tissue. (After Pierre Marie and Foix.)

"As regards their direction and their chances of meeting the bundles of the lower end, it may be said that it is relatively preserved. Even if there are several fibres tending to run a very oblique course, or even some few here and there which incline backwards, it may be said that the vast majority has kept to the longitudinal direction, and that most of them show

a sufficient degree of continuity to enable them to recover their original form.

FIG. 9.—Lateral notch. (Magnification, 7·5 diameters.)

E, notch. *n*, the nerve fibres are interrupted on the upper lip of the notch, where they form a small neuroma. *m*, showing nerve bundles travelling from end to end, myelinated in their upper part, and almost completely demyelinated in their lower part, which, however, is continuous. (After Pierre Marie and Foix.)

"In short, in the case of the pseudo-neuroma of

attrition even with a small lateral cicatrix, we think that the nerve should not be interfered with, no matter what the clinical syndrome may be."

The fairly frequent occurrence of a small lateral notch on one of the sides of the pseudo-neuroma should be noted (Pierre Marie and Charles Foix).[1]

3. **Lateral Notch.**—The cicatricial nodule often shows on one of its sides a notch which occasionally corresponds to an incomplete section; it can easily be seen at the part where the nerve is swollen (fig. 8).

When the notch is plainly visible to the naked eye the nerve often has two swellings, one above and one below the notch (P. Marie and Foix).

This is the lesion which Sicard and Jourdan have described under the name of nodules above and below the lesion.

From an histological point of view, the condition is the same as that described in the case of complete section; the wall of the notch itself is composed of very dense fibrous tissue.

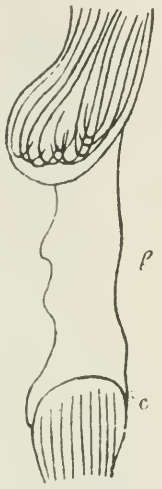


FIG. 10.

Section with pseudo-continuity.

a, upper end; *b*, irregular intermediate segment; *c*, lower end. (After Pierre Marie and Foix.)

With regard to the substance which joins the two swollen extremities of the nerve in the neighbourhood of the cicatrix, this is formed of fibrous tissue, which we have already mentioned, and in the more distant parts it shows the same histological characteristics as those seen in the pseudo-neuroma of attrition (fig. 9).

On the strength of these anatomical and pathological considerations, Pierre Marie and Foix advise that the surgeon should carefully preserve this bridge

[1] *Loc. cit.*

of nerve tissue, and should pare down the ends of the two swellings and the wall of the notch, and then suture end-to-end the lips of the wound in the nerve.

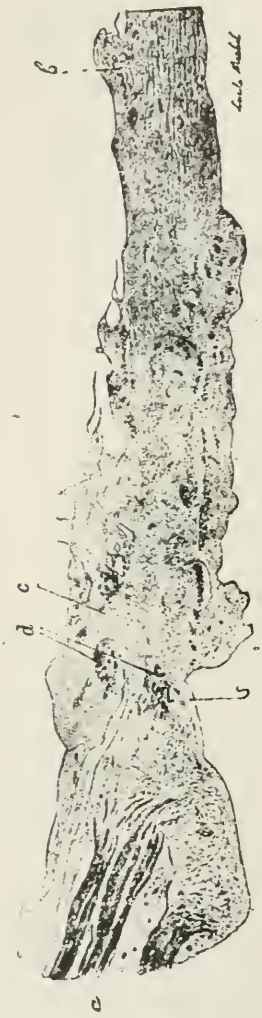


FIG. 11.—Pseudo-continuity.

a, nerve almost completely severed, forming a swelling with neuromatous tendency; *b*, lower end, which has partly preserved its normal arrangement; *c*, irregular intermediate portion; *d*, giant cells; *s*, point where the nerve has been divided. (After Pierre Marie and Foix.)

4. Accessory Types.—Among the lesions less frequently met with we will mention in the first place that of *pseudo-continuity*. Complete division of a nerve is an unusual occurrence, as we have already pointed out (from 20 to 25 per cent.); still more rare is the clean-cut section, with both ends far apart and out of line, which we mentioned in the first instance.

Anatomical section of a nerve occurs much oftener, in our opinion, in the form of pseudo-continuity (fig. 10).

Pierre Marie and Foix describe this lesion as follows: "The nerve shows two swellings at some distance from each other, of which the upper one is by far the larger. Between the two an apparent continuity is established by an irregular bridge, isolated by the dissection of the very dense fibrous tissue which filled up the space.

"Careful microscopical examination then reveals:—

"1. That the section is complete or almost complete.

"2. That the intervening bridge is too thin, long, and ragged, and has hardly any value from the point of view of regeneration.

"This bridge is always exclusively made up of fibrous tissue, to which is added a quantity of muscular tissue elements" (fig. 11).

This type of lesion, which is frequently met with in lesions of the musculo-spiral when the nerve is lost in the callosity of a fracture of the humerus, requires, from a surgical point of view, the wide resection of the fibrous bridge and the freshening up of the two ends of the nerve.

Lateral pseudo-neuroma, with or without a slight lateral notch.—This is the *lateral nodule* of Sicard and Jourdan, and the *eccentric* or *lateral cheloid* of M. and Mme. Dejerine. The swelling is irregular, and more in evidence on one side of the nerve (fig. 12).

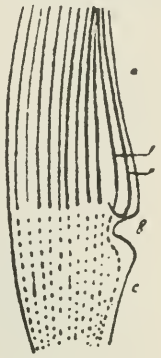


FIG. 12.

Lateral pseudo-neuroma with small lateral notch.

The pseudo-neuroma, which is more salient on the right side, shows at this level a small lateral notch. Of the fibres which were on their way to the upper lip, some turn back, thus forming a small neuroma, others curl in, pushed aside by the sclerotic tissue, and eventually join the lower end. (After Pierre Marie and Foix.)

Histologically, at the site of the lesion some nerve fibres are to be seen pushed back by the fibrous tissue, and others which had strayed from their useful direction (fig. 13).

Finally, there is a kind of lesion far less frequently met with than any of the preceding, the *small enucleable neuromata*, of an intra- or juxta-nervous character, *i.e.* either encapsuled within the nerve or jutting out on one of its sides (fig. 14).

These fibromata are of two kinds: the one are *pure neuromata*, formed of nervous fibres, and the others have a fibrous core. The first kind are situated in the interior of the nerve; they have a soft consistence, and should be left alone (fig. 15).

The second type form hard swellings on the surface of the nerve.

According to P. Marie and Foix, the best plan is to leave them alone, since the cicatricial tissue which replaces them when they have been excised is equally

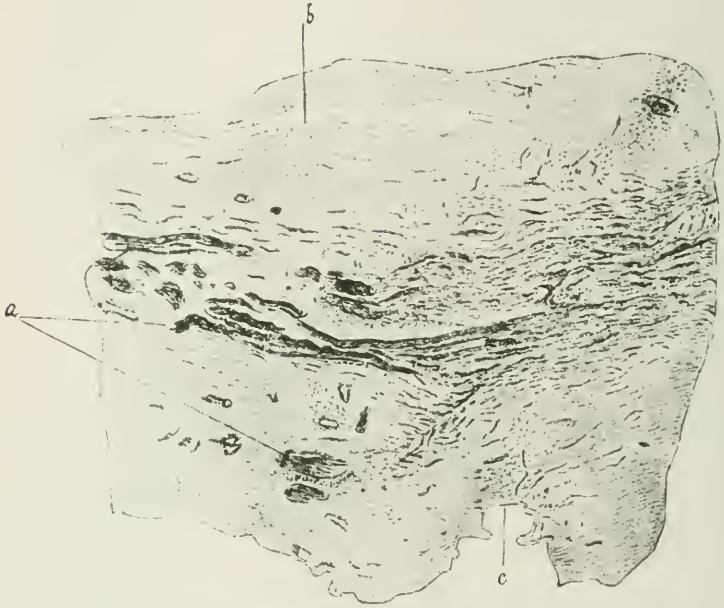


Fig. 13.—Lateral pseudo-neuroma with small lateral notch.

a, nerve fasciculus crossing the injured region ; *b*, sclerotic mass enclosing and thickening the nerve ; *c*, small notch which has determined the lateralisation of the pseudo-neuroma. (After Pierre Marie and Foix.)

hard, and occasionally presents an even greater obstacle to the passage of nerve fibres.

Simple induration of the nerve.—This lesion corresponds clinically in the vast majority of cases to those painful affections chiefly found in the median and sciatic, which M. and Mme. Dejerine and J. Mouzon call the " syndromes of irritation."

The nerve shows an induration which often can only be felt but occasionally can be seen, in which case the nerve is slightly swollen, and has a greyish tint,

or, on the contrary, may be congested and soft if the lesion is of recent occurrence.

Pierre Marie and Charles Foix have drawn attention to various inflammatory changes in their researches on the anatomical lesions of nerves, viz. interstitial neuritis with isolated areas of inflammation, parenchymatous neuritis, descending and retrograde changes in the axis-cylinders, the myelin sheaths and the structures of Schwann, gross vascular lesions, with very marked endarteritis of the medium-sized and small vessels, etc.

The existence of these lesions throws an interesting light on what must occur from an histological point of view in the case of simple induration of nerves, with painful symptoms of a causalgic character, trophic manifestations, and irritation of the motor fibres, etc.

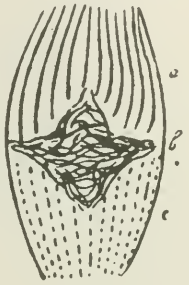


FIG. 14.

Small neuroma with fibrous core.

a, afferent fibres; *b*, other fibres; *c*, degenerated efferent fibres. (After Pierre Marie and Foix.)

Besides these lesions of the nerves proper we must note the existence of injuries to the tissues surrounding the nerve, to which Henri Claude, Vigouroux and R. Dumas have justly ascribed importance.

The fibro-sclerotic reaction is always very marked along the course of the projectile through the tissues, and increases when protracted suppuration has occurred in the wound, or when the injury to the nerve is serious, and is most marked when, in addition, a blood-vessel is wounded.

The exuberant lardaceous cicatricial tissue literally stifles what remains of the nerve cords and the blood-vessels.

Frequently the nerve becomes adherent to the blood-vessels and the muscles.

In addition, therefore, to the essential obstacles within the nerve itself which oppose its regenera-

tion, we must bear in mind that there are lesser impediments, but not the less real, engendered by the injuries to the surrounding tissues.

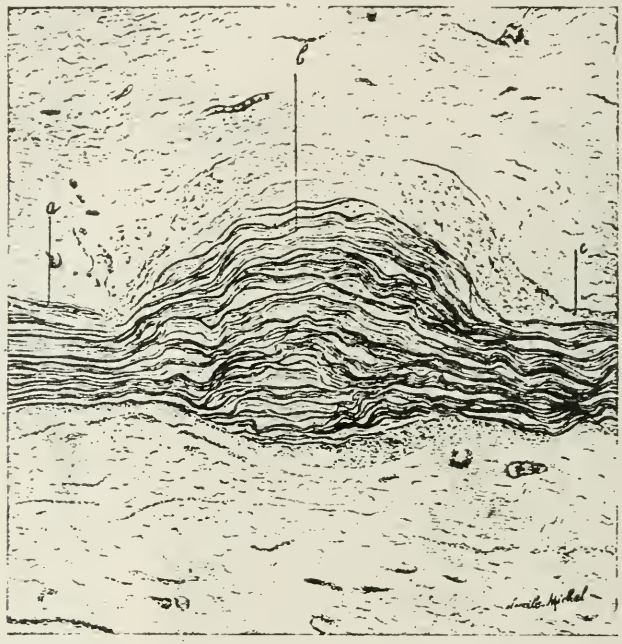


FIG. 15.—Small intra-nervous microscopic neuroma. (Camera lucida, obj. 4, oc. 1.)

The medullated fibres spread out in bundles, to join up again further on leaving the small neuroma. Some intra-nervous neuromata of larger size have the same structure. *a*, afferent end; *b*, neuromatous swelling; *c*, efferent end. (After Pierre Marie and Foix.)

CHAPTER II

RECOVERY OF SENSIBILITY

The English neurologist, Henry Head, performed a most interesting experiment in 1903. He caused his radial nerve to be divided and immediately sutured (the anterior branch and external cutaneous division in the neighbourhood of the elbow). In conjunction with W. H. Rivers he studied the nature and duration of the alteration of sensation as well as all the various stages during the recovery of sensation.[1]

Our observations, which were made on a large number of wounded, have shown us that in the large majority of cases the various stages in the restoration of sensation are identical with those described by Henry Head.

We shall therefore briefly state the findings of this neurologist, drawing attention in places to any instance in which we have encountered a different condition of things, and noting any special characteristic in the sensory disorders of other nerves than the radial.

In the first few days after the operation Head noticed the following facts, which we have been able to confirm in several cases of recent injury to the radial nerve:—

1. *Deep sensibility* in the radial area in the hand and forearm was *perfectly preserved.* Any moderate pressure in this region was well felt and localised. Passive movements of the muscles were fully appre-

[1] W. H. Rivers and H. Head, "A Human Experiment in Nerve Division," *Brain*, part iii., 1908.

ciated, as also was any movement of the joints; vibratory sensation was unimpaired.

2. *Superficial sensibility* was, on the contrary, *very much impaired* :—

(*a*) Touches with cotton-wool, or deformations of the skin produced by pulling the hairs, and even vigorous pinching of the skin, gave rise to no sensation in the area corresponding to the physiological sensory field of the radial nerve.

(*b*) Within the area of insensibility to superficial touch there was another zone totally insensitive to painful stimuli. On the border separating these two zones pricking was normally painful.

(*c*) Insensibility to *heat* and *cold* was absolute, even for temperatures above 50° C. and chilling produced by ethyl chloride spray.

The thermal changes occupied a zone which was generally more extensive than the area of analgesia, *without however exceeding the zone of insensibility to very light touch.*

(*d*) Although deep sensibility was preserved, *tactile discrimination* (circles of Weber) was greatly affected; the two points of a pair of compasses had to be separated by more than 8 centimetres in order to be recognised as two separate objects.

In the same way all appreciation of the size, shape, and dimension of objects was lost.

What was the subsequent course of this lesion, and what were the various stages in the course of sensory recovery ?

About fifty days after suture the extent of the analgesic area began to shrink, in addition cold was felt in certain parts of the zone still insensitive to cotton-wool.

At the end of four months sensibility to pricking and cold had still further improved, though heat was still not felt, except in the case of high temperature (50° C.), which caused a sensation of burning.

It was only after six months that even partial sensibility to superficial touch returned, due to pro-

gressive restoration of the sensitiveness of the hair; at the same time temperatures of about 45° C. were appreciated.

With the gradual return of sensibility to pain, cold and heat, was noticed *a tendency of the sensation to be widely diffused, to radiate to a remote part, and to become localised at a distance from the point of stimulation.*

These characteristics were especially well marked in the case of pricking and heat. When the skin was pricked in a part previously anæsthetic, sensation was delayed as compared to the normal side; at the same time the prick was more painful, although dull, ill defined, excessively unpleasant, and diffused, the painful sensation developing slowly, lasting for a long time, and unlike that felt normally as the result of a prick. Often the localisation of the pain was indefinite, and the subject described the point of pain at a considerable distance from the point actually stimulated.

At this stage of recovery sensibility is vague and incomplete. Head gives it the name of *protopathic sensibility.*[1]

About eight months after suture gentle stroking with a pledget of cotton-wool aroused a diffuse tingling sensation, ill localised, persistent, and often radiating.

This partial return of superficial tactile sensibility was due to the hairs, which became sensitive about three months after the operation. If the hairs were shaved, superficial sensibility was again abolished.

Within this area the interrupted electric current aroused no sensation.

Sensibility to cold returned earlier. Head attributes this fact to the existence of a greater number of "cold spots," *i.e.* of places in the skin specially sensitive to stimulation by cold. The "heat spots" must, according to Head, be fewer and more scattered.

It is only towards the four hundred and seventh

[1] W. H. Rivers and Henry Head, *loc. cit.*

day (fourteen months after section followed by suture) that a return of sensation was noticed for medium temperatures of about 37° C., accompanied by true appreciation of heat, which was then well localised.

From then onwards and for the next few months there was a gradual return both of superficial tactile sensibility (even after shaving the parts) and of sensibility to medium temperatures.

At the same time the tactile painful or thermal sensations lost their tendency to become diffuse, to radiate, to persist, or to manifest themselves in a disagreeable manner.

It was only very much later—two and a half to three years afterwards—that the skin regained its normal response even when shaved to quite superficial stimuli, and that the two points of Weber's compasses were clearly felt separately, and that the size and shape of objects were appreciated as well as temperatures between 26° and 37° C.

These secondary elements of sensory recovery constitute Head's *epicritic sensibility*, which eventually completes the protopathic sensibility.

These are the main points which the English neurologist brought out in his own case, in which the nerve had been divided and sutured under aseptic precautions.

In the course of recovery of sensation in nerve lesions due to war wounds we consider the earliest sign of recovery to be *pain when the skin is pinched within the sensory area of the injured nerve.*

This sign has been noted and described by André Thomas[1] and Belenki,[2] who considers it important, enabling one to recognise that the injured nerve is

[1] André Thomas, "La sensibilité douloureuse de la peau à la piqûre et au pincement dans la période de restauration des nerfs sectionnés après suture ou greffe," *Soc. de Neur.*, Feb. 3, 1916. *Revue Neurologique*, Feb. 1916, p. 311.

[2] Belenki, "Les symptômes sensitifs dans les sections anatomiques et physiologiques des nerfs périphériques," *Presse médicale*, Feb. 17, 1916.

still connected with the sensory centres. A. Thomas rightly draws attention to the fact that this sign long precedes the appearance of areas which are parænsthetic on pricking.

Pain on pressure of the nerve-trunk below the site of injury and the "formication of recovery" are later manifestations.

This ill-localised, unpleasant, and persistent pain produced by pinching the skin appears to be localised in small circumscribed spots, just as the paræsthetic zones are later on for pricking and heat (A. Thomas).

It appeared to us that these areas occupied mainly the middle third of the posterior aspect of the forearm in the case of injury to the musculo-spiral, and the lower third of the outer aspect of the leg in the case of injury to the great sciatic, and more especially to the external popliteal.

In the case of lesions of the ulnar, the seat of election is the inner aspect of the hypothenar eminence. These zones soon spread and coalesce, finally involving the whole of the sensory area of the nerve in question. They are at that time insensitive to pricking and to high temperature (45°).

This phenomenon is explained by Thomas in the following way :—

"Between the time when the sensory fibre reaches the dermis and that at which it spreads out into the terminal organs or the epidermis, the return of sensibility undergoes a delay comparable to that of motor fibres in the interval which separates their entrance into the muscle and their adaptation to the motor end-plates.

"It is not surprising that pinching should be felt before pricking. When the manipulation includes the whole skin and the subcutaneous cellular tissue, it affects the nerves themselves and the subdermic organs; when it involves the skin only, it affects the terminal organs and a certain number of fibres which may not have yet crossed the deepest layers of the epidermis; it thus reaches fibres which the more

superficial pricks cannot affect, and it reaches a greater number of them." [1]

This sign appeared to us to precede the appearance of *formication*, noted by Létievent and recently investigated and described by Tinel.[2]

Formication, which the patients compare to the sensation of an electric current, or more or less disagreeable tinglings, occurs in a large number of wounded, during convalescence, after a severe lesion or nerve section with subsequent suture. It makes its appearance several days after nerve sutures when the division of the nerve is of recent date.

The patients state that a slight tap with the finger on the cicatrix of the wound, especially over the probable seat of the nerve lesion, elicits a kind of electric vibration, coursing throughout the whole limb, but radiating mainly in a certain part or over the whole of the sensory area of the nerve.

To appreciate the value of this sign, frequently repeated examinations at close intervals of the character of this formication are necessary, as urged by Tinel and André Thomas.

We have just seen that the first appearance of formication is noticed very soon after nerve suture; nevertheless, so long as formication is only elicited by pressure or percussion of the nerve at the cicatrix, it does not indicate that the axis-cylinders of the central end have reached the peripheral end, but rather suggests that a certain number of nerve fibres, if not all, have encountered an obstacle, and that they have been rolled up inside a neuroma of such a size that frequently it can be definitely palpated. A few of these fibres have strayed into the neighbouring tissues; any irritation of the cutaneous cicatrix, such as pricking or stroking with a brush, is felt not only at the actual place of stimulation, but also at the periphery of the limb, in the area supplied by the

[1] *Revue Neurologique*, Feb. 1916, pp. 312–313.

[2] Tinel, "Le signe du 'fourmillement' dans les lésions des nerfs," *Presse médicale*, Oct. 7, 1915.

branching out of the sensory fibres of the nerve (cicatricial topoparæsthesia).[1]

If repeated and successive examinations continually confirm the existence of this neuroma, or swelling, in the course of the nerve, which is painful on palpation, and pressure on which is accompanied by radiating sensations of a more or less disagreeable nature referred to the periphery of the nerve, this sign must not be interpreted as the initiation of recovery, but should rather be taken to suggest the interposition of an obstacle to regeneration of a part or the whole of the sensory fibres of the nerve.

If, on the contrary, *palpation and pressure on the nerve below the site of the wound give rise to formication, and if, in addition, this pressure is painful*, there is every reason to assume that neurotisation is taking place in the lower end.

In fact, while the sensation of formication thus descends along the course of the nerve, *pain on palpation of the nerve-trunk itself appears*, and this pain increases as the site of the lesion is approached. During this time the pain felt on pinching the skin becomes more intense, and its radiation towards the extremity of the limb increases.

A short time after the appearance of these signs the wounded complain of *pain becoming more and more defined*, mainly during the time they are massaged, *in the muscles whose nerve-supply is given off highest from the injured nerve* ; such is the character of the pain felt in the supinator longus in the neighbourhood of its upper insertion, or in the gastrocnemius and soleus near the popliteal space.

This pain, which is at times very intense, similar to that noticed in certain painful affection of the nerves, has a complex pathogeny. It may be due to the sensory fibres having been directed to the muscles " by a sort of pointsman's error " (A. Thomas), or to the irritation of the nerve-trunk itself and of its

[1] André Thomas, *Société de Neurologie*, April 6, 1916. *Revue Neurologique.*

sensory filaments, an irritation which often develops during the process of recovery.

With regard to the pathogeny of formication, Tinel attributes it to the advance of the regenerated axis-cylinders into the peripheral end of the nerve.

The signs which we have just described constitute the **first manifestations of sensory recovery.**

They invariably precede the return of electrical contractility, muscular tonus, and voluntary movement.

Henceforward recovery progresses along the lines described by H. Head.

First of all, *sensibility to a prick* reappears in the zone immediately adjoining the areas which in the first instance were quite irresponsive to any stimulation. This sensibility is at the start defective, or, to use Head's term, "protopathic."

Pricking is felt some time after contact over a wide area, and is interpreted as a vague, diffuse, persisting pain, radiating to remote parts, nearly always in the same directions. Thus in one of our patients, with a wound of the great sciatic, a prick at a point 10 centimetres above the tip of the external malleolus radiated, after several seconds' delay, into the heel and sole of the foot; a prick over the external malleolus itself radiated to the external border and dorsum of the foot. These facts are ingeniously explained by Thomas as *errors in the switching* of the sensory fibres.

While this return of sensibility to pricking is developing and becoming more definite, thermal sensibility is beginning to reappear. *Ice and very hot water* are appreciated, with great delay at the borders of the area which was primarily anæsthetic. But the sensations are still vague, and often the patient is in error in his replies as to the temperature.

Medium temperatures (between 15° and 37° C.) are only distinguished very much later, towards the end of recovery, when all forms of sensibility have returned.

Moderate cold is felt earlier, and is more correctly interpreted, than *tepid heat*, which, at any rate in certain circumscribed areas, is always misjudged,

even when motor, sensory, and electrical recovery is complete.

Along with the appearance of islands of paræsthesia to pricking and heat we see the return of *sensibility to a superficial touch.* This form of sensibility returns late, but once it has returned it recuperates more rapidly and more completely than the sensation for pricking, which requires a long time to regain its normal activity without the accompaniment of diffuse and radiating pain.

Head noted that this superficial tactile sensibility, which returned several months after suture, disappeared again when the areas originally anæsthetic were shaved.

It is necessary to realise that, even in perfect health, all nervous lesions being excluded, superficial tactile sensibility diminishes in delicacy as soon as the parts are shaved. From this point of view Belenki rightly distinguishes between the hair sensibility and cutaneous sensibility proper, and insists on the importance of eliciting this latter by a piece of paper or cotton-wool, since superficial tactile sensibility is far less acute in the parts naturally bald than in those normally covered with hair.

Deep sensibility (deep pressure, vibrating sensation, and recognition of position) once abolished, as in lesions of the ulnar, median, and the great sciatic nerves, reappears much later. It is as a rule regenerated by the time superficial " epicritic " sensation has returned, *i.e. the return of tactile discrimination* (compass points of Weber), *recognition of medium temperatures, and precise identification of objects* (stereognostic sense).

During the first period of recovery of deep sensibility, possibly on account of the defective superficial sensibility,[1] patients feel as if their limbs, or parts

[1] The return of hair sensibility (traction of hair) in the first place, and later the perception of superficial touch, are accompanied at first, as has been noted by Head, by indefinite and diffuse tingling, which persists and radiates to a distance.

It is only at the time of the return of epicritic sensibility that very superficial and light tactile sensations are correctly appreciated.

of their limbs which were previously anæsthetic, were inclosed in a vice. This is the case with patients suffering from injuries to the sciatic, in whom investigations of this kind are most interesting.

After these general considerations let us examine shortly each of the important nerves of the limbs in regard to the interesting peculiarities of their sensory disturbance and recovery.

Musculo-spiral Nerve.—We have on many occasions laid stress on the variability and the slight degree of sensory changes in lesions of the musculo-spiral nerve.

In several cases of recent complete division of the musculo-spiral one occasionally notices an anæsthetic zone embracing a wide expanse of the physiological sensory area of the radial in the hand, as in Head's experiment.

In other cases, comprising severe injuries or anatomical section, complete division dating back several months (in many of our cases the division of the nerve had occurred about one year before), sensory disturbances are far less marked. In particular, anæsthesia to superficial touch affects only certain circumscribed areas on the dorsal aspect of the thumb, the skin over the metacarpo-phalangeal joints of the index and middle finger, and a small area in the first dorsal interosseous space quite close to the fold of skin uniting the thumb and index.

In the rest of the territory supplied by the musculo-spiral, touch is normally felt (especially is this the case in those parts provided with fine hair). The same applies to moderately deep pricking. More superficial pricking is often interpreted as a touch, but is usually well localised.

Heat and cold are, on the contrary, for a long time mistaken for tactile sensations. Cold is generally better appreciated than heat, but with delay; high temperatures of 45° to 50° C. are felt with a certain delay, and as a burning pain.

Tactile discrimination (circles of Weber) is often

defective; the points of the compasses have to be separated from 6 to 7 centimetres to be felt separately, and generally it is only the upper point that is localised by the patient.

What are we to think of those cases where, despite complete division of the nerve, the sensory nervous manifestations are unimportant, and appear to diminish gradually without there being on the other hand any definite signs of recovery ?

It is generally admitted [1] that a supplementary nerve-supply from anastomoses occurs in the first few days following the physiological interruption, but that eventually disturbance of sensation always occurs when there is no regeneration or neurotisation of the distal part of the nerve. This is one of the signs of the " syndrome of complete interruption " described by M. and Mme. Dejerine and J. Mouzon.

In practice it is probable that this supplementary nervous supply undertaken by the neighbouring nerves can establish itself for a fairly long period, or else one is forced to admit that in the majority of cases the sensory area of the hand supplied by the radial is very restricted, and that the musculo-cutaneous on the one hand and the median on the other takes its place not only as regards deep sensibility, but as regards superficial sensibility as well.

As for the sensory area of the radial in the forearm, the types of disturbances that may be found there are still more ephemeral and changing.

The greatest change is towards the perception of pricking, cold, and particularly heat.

At the commencement of the recovery of a severe injury to the musculo-spiral, if the skin is pinched up between finger and thumb in the region normally supplied with sensation by the nerve in the forearm, intense pain is elicited of a diffuse and burning character which does not occur in the normal limb. This pain often increases in intensity when the distal extremity of the limb is approached.

[1] M. and Mme. Dejerine and J. Mouzon, *Presse médicale*, May 10, 1915.

In the region of the anterior branch to the hand the pain on pinching, although well felt when recovery is advanced, always seems less marked than when the forearm is pinched.

Formication must be elicited by deep palpation of the nerve in the musculo-spiral groove, and especially at the neck of the radius.

In the case of transmission of nervous currents, the sensation of the electric current radiates into the first dorsal interosseous space and on to the dorsum of the thumb.

Median Nerve.—In severe wounds of the *median*, anæsthesia, as already stated, affects the last two phalanges of the index and middle fingers. This anæsthesia is complete to all modes of sensation, both superficial and deep, since the median supplies both superficial and deep layers of this region.

To test superficial tactile sensibility a more powerful stimulus than that exerted by the wisp of cotton-wool will be necessary, for in the normal state the skin of the palm of the hand is insensitive to light stimulation by cotton-wool, just as it is less sensitive than the back of the hand to thermal stimulation.

It is well, therefore, not to conclude that there is tactile anæsthesia of the palmar surface of the thumb and thenar eminence because these regions are insensitive to very light touching by cotton-wool.

In the course of lesions of the median, sensory recovery generally antedates the return of volitional motor power, but it remains defective for a long time in a stage of paræsthesia and in a protopathic condition. The re-establishment of electrical contractility and the appearance of voluntary movements take place before tactile discrimination, stereognostic sense, and the appreciation of medium temperatures are sufficiently developed.

The median nerve, more than any other nerve in the arm, gives rise, during the time of its sensory recovery, to painful sensations, which are either spontaneous or elicited by pressure on the nerve-trunk

below the seat of the lesion (path of the nerve in the forearm, thenar eminence). These manifestations recall in a measure what occurs in certain painful wounds of this nerve.

Pain when the skin is pinched is easily investigated on the thenar eminence, the palmar aspect of the thumb, and of the basal phalanges of the index and middle finger.

To elicit formication the nerve must be compressed below the lesion, on the inside of the arm, the middle of the bend of the elbow, or on a line drawn along the middle of the anterior surface of the forearm. This sensation radiates to the palm of the hand and to the extremity of the index and middle finger.

FIG. 16.—Sensory changes after injury to the ulnar nerve in course of recovery.

Black: anæsthetic area. *Grey*: hypæsthetic area. *Dotted*: area of paræsthesia (diffused and persistent pain on pinching the skin and on deep pricking).

The Ulnar Nerve.—The *ulnar nerve* possesses like the median a sensory area of its own, which occupies the little finger and the innermost part of the hypothenar eminence. Anæsthesia is complete in this region when the nerve is injured.

When the nerve is recovering, a fairly severe pain is felt if the skin of the hypothenar eminence is pinched. This pain radiates to the little finger and the forearm, and, together with the sensation of formication, may extend up to the extremity of the little finger, as well as pain on pressure of the hypothenar muscles (fig. 16).

Great Sciatic Nerve.—During the first stage of sensory recovery pain is noticed on pinching the skin in the region supplied by the peroneal nerve, a branch of the external popliteal, the skin on the outer part

of the leg, which is coarser, being very tender to pinching. Later on medium pricking and heat cause pain, which radiates into certain areas according to the points irritated (fig. 17).

At the same time formication produced by pressure upon the trunk of the external popliteal over the neck of the fibula and the musculo-cutaneous nerve becomes more accentuated, and radiates towards the dorsum of the foot.

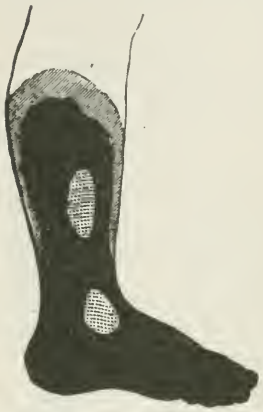


Fig. 17. — Distribution of sensory changes in a lesion of the great sciatic in course of recovery.

Black: absolute anæsthesia. *Grey*: hypæsthesia. *Dotted*: areas of paræsthesia (deep pricking and heat (45° C.) elicit diffuse and severe persistent pain, felt more definitely in the back and sole of the foot than at the points of stimulation).

Spontaneous aching in the muscles and pain on pressure of the gastrocnemii become predominant signs.

In the foot, the appearance of the areas of paræsthesia is more delayed. It is the outer border of the foot (external saphenous) which first recovers some sensibility to deep pressure.

Deep sensibility in the foot as well as superficial sensibility requires a very long time to reappear (sole and toes). It is only starting at the time when a large number of muscles have regained their power of voluntary movement and electrical contractility.

External Popliteal.—In the case of an isolated lesion of the external popliteal sensory disturbances are less marked, in particular the deep sensibility of the dorsum of the foot is little affected.

Its sensory recovery is more rapid, and progresses according to the order just laid down for the great sciatic. The external popliteal has from every point of view much resemblance with the musculo-spiral, whereas the internal popliteal behaves like the median.

CHAPTER III

MODES OF RECOVERY OF ELECTRICAL EXCITABILITY IN THE COURSE OF NERVE INJURIES

Little importance was attached at the beginning of the war to the study of the diagnostic and prognostic value of electrical examination, although the large number of injuries to peripheral nerves offered a vast field for such researches.

At first electro-diagnosis roused much enthusiasm among neurologists and surgeons, the latter hoping to replace all clinical examinations by a rapid electrical examination, which was at one and the same time to reveal which nerve was affected, and the situation, nature, and extent of the lesion (section, crushing, compression, or simple irritation).

Very soon came disillusionment ; complete division of the nerves was found in cases in which electro-diagnosis indicated partial R. D., and in other cases in which the R. D. was well-marked there was occasionally found to be no solution of continuity of the nerve, or it appeared even to be intact.

Henceforwards electro-diagnosis was discredited, and declared to be suitable only for detecting paralyses due to psychical, functional, or other causes.

Specialists took part in the discussion in order to define the true characteristics of the R. D. and its significance from a diagnostic and prognostic point of view. They rightly insisted that the R. D. syn-

drome is always changing and provisional, and that it requires a correct interpretation.[1]

Electrological findings arrived at previous to the war remain in general perfectly correct.

We shall first give a short account of the prevalent views as to the occurrence, characteristics, and changes in R. D., and then describe some phenomena which have struck us in the course of the very numerous electrical examinations which we have had occasion to perform.

Let us first state that the method we used was of the simplest. We conducted the investigations with the electrical apparatus of the most elementary description with which every clinician can provide himself, and it is with this that all our examinations were made.

Faradic investigations were made with an induction coil with two dry cells (Gaiffe's model). We have nearly always used the bipolar method of Duchenne (of Boulogne), and only occasionally the unipolar method. The electrodes consisted of either two egg-shaped bulbs or of the two round button-shaped pads familiar to every one; we used the current supplied by the coil wound with fine wire, the maximum intensity being obtained by the two coils being wholly engaged.

For galvanic investigations we used a battery consisting of thirty-two cells, with the indifferent electrode applied between the shoulder blades or in the lumbar region, according to which limbs were being examined, and an active electrode consisting of a pad about $2\frac{1}{2}$ centimetres in thickness. We have not used the current discharged from a condenser nor any other method requiring complicated instruments. The cases we examined had received their wounds some time previous, at least six weeks beforehand.

Characteristics of the Reaction of Degeneration.—The reaction of degeneration, which is very frequently seen as soon as the nerve is sufficiently affected to

[1] A. Zimmern, *Éléments d'Électrothérapie clinique*, 1906. Published by Masson.

show any marked paralysis, is the rule in cases of total paralysis.

At first it consists in *abolition of the faradic and galvanic excitability of the nerve-trunk. The paralysed muscles do not react to stimulation by the faradic current, and their reaction to stimulation by the galvanic current is lessened and altered in character* (slow contraction, inversion, or equalisation of the polar formula). The most constant qualitative modification is the *slowness of contraction*, which is always sufficiently marked to be appreciated by the naked eye: the other modifications are less regularly seen, such as *polar reversal* (increase of muscular contraction or more rapid reaction to closure of the anode than to closure of the cathode) or *polar equality* (K.C.C.=A.C.C.); both these latter are nevertheless important characteristics of R. D.

An indispensable characteristic, and one to which we attach the greatest importance, is the *longitudinal reaction*, to which Huet has drawn attention.[1]

It is elicited by stimulating the lowest part of the muscle near its tendon, so that the current passes throughout the length of the muscle. In cases of nerve lesion with R. D. the longitudinal reaction, which, according to the text-books, is more marked than that obtained by stimulation of the points of election, is also accompanied by a slow contraction. Later on the longitudinal hyperexcitability is only relative, and in order to demonstrate it fairly strong currents are necessary.

During R. D. the longitudinal reaction is accompanied as a rule by a more marked cathodal than anodal contraction; much more rarely the contraction predominates at the anode.

Such is the description of the reaction of degeneration according to the generally accepted view. We must now consider how it develops, increases, and disappears.

[1] E. Huet, "Quelques considérations sur l'électrodiagnostic," *Bulletin de la Société d'Électrothérapie et de Radiologie*, April 1915.

The generally accepted ideas, confirmed by specialists in the course of their observations on war wounds, are as follows :—

Appearance of R. D.—The initial phase of R. D. is characterised by [1]—

1. *Progressive diminution of excitability of the nerve to both the faradic and galvanic current.*

2. *Progressive diminution of faradic excitability of the muscle.*

3. *Galvanic hyperexcitability of the muscles.* The period occupied by this phenomenon is often fairly short, at other times it may last several months.

4. *A tendency to sluggishness in the contractile response of the muscle.*

The changes in electrical contractility commence four to six days after the injury, as soon as degeneration of the lower end starts to assert itself.

This preliminary phase lasts about two weeks. R. D. is then established, and is manifested by the characteristics which we have described.

After several weeks, or even months, some of these characteristics change. A lowered excitability to the galvanic current succeeds the hyperexcitability seen at the outset, the other signs of R. D. persist, *e.g.* sluggish contraction, polar reversal, and liveliness of the longitudinal reaction, compared with the response elicited from the motor points. This stage may last for months, or even years, according to some observers.

Evolution of R. D.—If the lesion of the nerve is irreparable (complete section without regeneration), the hypoexcitability of the muscles continues to become more pronounced, and ends, in the case of complete atrophy and degeneration of the muscles, in muscular inexcitability to stimulation, both at the motor points and in the tendons, but the longitudinal reaction is the last to be lost.

[1] A. Zimmern, "Quels enseignements nous fournit la réaction de degénérescence dans les blessures des nerfs," *Presse médicale*, April 15, 1915.

On the other hand, when the lesion is undergoing repair, excitability of the nerves to both the faradic and galvanic current reappears, and subsequently the muscles in their turn respond to stimulation. The *return of faradic excitability of the muscles does not occur*, according to the generally accepted view, *until a certain time after the nerves have recovered their excitability to stimulation.*

Qualitative defects in galvanic excitability are modified in their turn, in that the contraction becomes less sluggish, the contraction of the muscles grows more and more active, and the excitability at the motor points becomes more marked than the longitudinal reaction. Polar reversal persists for a considerable time as a reminder of a previous lesion.

During this progress of R. D. towards the normal, voluntary motor power has become re-established. *This re-establishment of voluntary motor power precedes by some time*, according to the classical opinion of Duchenne of Boulogne, *the return of the faradic excitability of nerves and muscles.*

Finally, when all qualitative changes caused by the R. D. have disappeared, there still persists a general condition of hypoexcitability for faradic, and especially galvanic, stimulation, more marked in general in the muscles than in the nerve-trunks.

Such, then, is the evolution of the *complete R. D.* following division or severe injury of a nerve with interruption of its physiological function.

When the lesion is less severe, complete R. D. is replaced by a *partial R. D.*, which consists in—

1. Diminution of faradic and galvanic excitability of the nerves.
2. *Diminution of faradic excitability of the muscles.*
3. Qualitative impairment of galvanic excitability (sluggish contraction, predominance of the longitudinal reaction, reversal or equalisation of the polar formula, or persistence of the normal formula (NF>PF).

In the light of the foregoing, the interpretation of

the observations made on the wounded in this war will be possible.

Although anatomical division of nerves is rare, interruptions of the physiological functions of the nerves, on the other hand, are of frequent occurrence. We have not had the opportunity of seeing a sufficient number of recently wounded men; for this reason we are unable to say whether hyperexcitability of the muscles to galvanism is a constant sign, nor can we say at what point of time exactly it appears or wanes.

The following is a *résumé* of the electro-diagnosis carried out on two cases which we were able to examine a very short time after they were wounded :—

Case I.—Jules L——, wounded May 7, 1915, by a shrapnel bullet high in the upper arm.

Total paralysis of the musculo-spiral, paresis of the biceps, and pain in the region supplied by the musculo-cutaneous in the forearm and hand.

Electrical examination for the first time May 12 (five days after being wounded).

Faradic Current.—Inexcitability of the musculo-spiral nerve. Hypoexcitability of the paralysed muscles, with wave-like fascicular contractions. A very strong current was necessary to cause the tendons to rise.

Galvanic Current.—Musculo-spiral nerve inexcitable. *Very marked hypoexcitability of the paralysed muscles* without sluggishness of contraction, but with a tendency to equalisation of the polar formula.

Examination on May 15, 1915 (eight days after being wounded).

Faradic Current.—Inexcitability of the musculo-spiral nerve in the arm. Very marked hypoexcitability of the paralysed muscles.

Galvanic Current.—Inexcitability of the musculo-spiral nerve. Hypoexcitability and polar reversal for the biceps.

Very marked hypoexcitability of the musculo-spiral muscles, but without sluggish contraction.

Examination on May 17, 1915 (ten days after being wounded). Faradic hypoexcitability of the muscles supplied by the musculo-spiral and of the biceps; galvanic inexcitability of the same muscles.

In all subsequent examinations faradic excitability never quite disappeared.

The paralysis decreased as time went on, so that at ten months after the wound it can be considered as cured.

When examined on December 6, 1915, the faradic hypoexcitability of the muscles had diminished, the nerve was excitable by the interrupted current, and galvanic inexcitability had been replaced by galvanic hypoexcitability coupled with polar reversal, but without any sluggishness of the contraction.

CASE II.—Captain C——, wounded August 24, 1915. Lesion of the right brachial plexus (muscles supplied by the musculo-spiral, flexors of the fingers, thenar and hypothenar muscles, and interossei) and of the axillary artery.

First electrical examination September 13, 1915 (twenty days after receiving the wound).

Faradic Current.—Inexcitability of the musculo-spiral nerve.

Hypoexcitability of the ulnar and median nerves.

Hypoexcitability of the muscles supplied by the musculo-spiral nerve, and of the thenar and hypothenar muscles.

Galvanic Current.—Sluggish contractions in the same muscles.

Examined on September 21, 1915 (about one month after being wounded).

Faradic Current.—Hypoexcitability marked in the triceps and thenar muscles, less marked in the other muscles supplied by the musculo-spiral, and in the flexors and hypothenar muscles.

Galvanic Current.—Excessive hypoexcitability and slight sluggishness of the contraction in the musculo-spiral muscles, the thenar and hypothenar muscles, and interossei.

Paralysis has steadily diminished.

In March 1916 (seven months after being wounded) the extensors of the hand and wrist have in a great measure recovered, as also the flexors and the interossei. There is still marked paralysis of the triceps, abductor longus pollicis, and thenar muscles.

Thus in these two cases faradic excitability had not completely disappeared, and paralysis had diminished. The impairment to galvanic excitability was purely quantitative in the first case, but assumed the characteristics of R. D. in the second case. The mild nature of the first case well confirms the fact to which Huet has drawn attention—namely, that the return to volitional motility occurs far more promptly in those muscles showing very marked hypoexcitability than in other muscles exhibiting the usual characteristics of the R. D.

In serious lesions due to wounds the injured nerve and the muscles supplied by it are inexcitable on faradic stimulation.

There is galvanic inexcitability of the nerve, often coupled with considerable hypoexcitability of the muscles, sluggishness of contraction, polar reversal

for certain muscles, and, on the contrary, equalisation or a normal polar formula for other muscles.

Longitudinal excitability from the first few weeks onwards after injury exceeds the reaction obtained at the motor points. We have never been able to find in patients who have been wounded at least six weeks previously any hyperexcitability of the muscles with the galvanic current, either when there was total anatomical section, or in those cases where the interruption was of a physiological nature of long standing.

In these cases we have always found faradic inexcitability of the paralysed muscles three months after the infliction of the wound.

Among those with wounds of the musculo-spiral or sciatic whom we were able to follow up systematically,[1] some showed complete section or serious injuries, which necessitated suturing or resection; others, who were not operated on, were so severely injured that they showed only slight signs of return of sensibility even after a lapse of six months.

Among the first lot (those operated on) we noted *a return of faradic contractility* from the third week to the fourth month after operation.

It was necessary to use a strong current, obtained by totally engaging the coil wound with fine wire, and the electrodes had often to be applied below the motor points.

The contraction was feeble, occasionally quite localised, but sometimes it was transmitted down to the tendon, and a corresponding movement was elicited. Positive results were chiefly obtained in those muscles which were easily accessible, such as the tibialis anticus, peronei, gastrocnemii, extensor longus digitorum, extensor proprius hallucis, radial muscles, supinator longus, extensor communis digitorum, thenar and hypothenar muscles, and interossei.

[1] In investigating the course of electrical reactions it is easier to study musculo-spiral paralysis or lesions of the great sciatic and external popliteal, since the phenomena of diffusion are easier to interpret in these cases.

We reported eleven of these cases to the "Société de Neurologie,"[1] viz. :—

(*a*) Two complete sections (sciatic and external popliteal) operated on two months after being wounded (end-to-end suture); sixty days after operation faradic contractility appeared.

(*b*) One resection (external popliteal) performed four months after being wounded; twenty-two days after operation faradic contractility reappeared.

(*c*) One partial resection (external popliteal) performed six weeks after being wounded; seventy-five days after operation faradic contractility reappeared.

(*d*) Three hersages (great sciatic severely injured) performed four and seven months after the wound; sixty days and one hundred days after operation faradic contractility reappeared.

(*e*) Four freeings (one great sciatic, one external popliteal, and two musculo-spiral cases); faradic contractility reappeared in from two to three months after operation, and six to eight months after receipt of injury.

The cases progressed as follows :—

(*a*) One of the two complete sections in which suture was performed by M. Gosset under perfect conditions does not show any return of voluntary motor power twenty-one months after the injury (nineteen months after operation). There are some indications of recovery of sensation. Certain muscles continue to react to faradic stimulation. In the second case of complete section there is return of voluntary motility in the tibialis anticus one year after operation and fourteen months after injury.

(*b*) Total resection has resulted at the end of six months in restoration of voluntary motility, which is still improving.

(*c*) Partial resection has had a similar course.

[1] Pierre Marie and Mme. Ath.-Benisty, "Du retour de la contractilité faradique avant le rétablissement de la motilité volontaire dans les muscles paralysés, à la suite des lésions des nerfs périphériques," *Société de Neurologie de Paris*, April 15, 1915, and May 6, 1915. *Revue Neurologique*, May–June 1915, p. 494, and July 1915, p. 557.

(*d*) Of the three hersages of the great sciatic, two have been followed by progressive motor restoration, the third shows the same slight degree of faradic contractility without any appreciable return of voluntary motility one year after operation.

(*e*) Finally, the four freeings have resulted in three cures; the fourth (great sciatic trunk in the region of the buttock) has not shown any improvement, and we have also lost sight of this patient.

From all this, it is justifiable to conclude that this partial return of faradic excitability should be interpreted as a prognostically favourable sign.[1]

Of course in many cases of severe wounds (sections with suture or regeneration after resection) we witnessed the confirmation of the generally-accepted rule that voluntary motility was restored, at any rate to a considerable extent, even at a time when electrical contractility, and especially faradic excitability, still remained in abeyance.

But in a large number of cases we have seen *faradic contractility reappear, partly at any rate, several weeks, or even months, before voluntary motility appeared.* This often coincides with sensory recovery (formication, deep aching in the muscles, or paræsthesia).

What can we conclude from these facts?

Every time there has not been section or resection of a nerve one should ask oneself if faradic excitability has ever totally disappeared. Nevertheless the electro-diagnosis of all our wounded has always been carried out in a most thorough manner, and repeated many times by specialists.

We have often found when examining nerve lesions in course of recovery that there is no suggestion of faradic excitability in the muscles, although they had reacted definitely two or three days previously, and did so again later. Occasionally the mere inversion of the poles was sufficient to determine a fresh contraction.

[1] Since these two reports we have been able to find this early return of faradic contractility in a large number of cases. These signs have invariably been of good import.

The most surprising thing was to see this disappearance of faradic excitability coincide several times with a return of voluntary motor contraction. The current then diffused itself into the neighbouring healthy muscles.

Moreover, in several of these cases, not only did the faradic excitability disappear, but the galvanic as well. One could no longer obtain either the longitudinal reaction or the sluggish contraction which had existed throughout the whole period of the paralysis. Excessive diffusion, even with a feeble current, occurred into the sound muscles. We have observed several instances of this sort in typical cases where voluntary motility reappeared before faradic contractility.[1]

We may also mention that *we have not always seen the assertion confirmed that faradic excitability of the nerve re-establishes itself before faradic excitability of the muscle.* In many of the cases where partial faradic excitability of the muscles took place, the nerve remained inexcitable by transdermic methods. The importance of this phenomenon might be denied by attributing it, and quite justifiably, to a certain difficulty in reaching the nerves through the superficial structures, to the resistance of the tissues, a sudden diminution in the intensity of the faradic current, which, though difficult to detect, is quite possible, or even to a polarisation of the electrodes.

Nevertheless, in a case of musculo-spiral paralysis of three months' standing, in which the muscles reacted very satisfactorily to the faradic current, we found in the course of an operation (liberation of the nerve) that the nerve-trunk directly stimulated in the operation wound hardly reacted to a current of equal force. The electrodes, it is true, were different.[2] But the same current applied to the paralysed muscles

[1] All our investigations on faradic contractility of the paralysed nerves were conducted by the bipolar method; the unipolar method and the tetanising current did not give any appreciable results.

[2] The electrodes used in this instance were specially sterilisable electrodes devised by H. Meige.

which had been exposed produced fairly powerful contractions in accordance with the excitability tested by the percutaneous method.

In another case (paralysis of the musculo-cutaneous) direct faradic stimulation of the nerve was negative even when the muscle of the biceps was feebly excitable.

To change the subject, and to return to the two cases of complete section sutured under very satisfactory conditions, which we referred to previously, it may be asked why, without any return of voluntary motility in one case, or with partial or late return in the other, faradic excitability of the muscles still exists? This excitability varies, it is true, from day to day; it is only slight, but still it has persisted now for more than twenty months. One of two things has happened: either the suture of the nerve has been a failure, and fibrous tissue has been interposed between the two ends, impeding all advance of the axis-cylinders towards the lower end, and division persists for this reason. In that case it is not clear why the muscles have not also become completely degenerated.

The other explanation is, that the suture has helped in recovery, which is manifested by sensory symptoms and by a return to faradic excitability in the muscles.

In cases of anatomical section not repaired by suture, or of crushing or compression by callus, faradic and galvanic excitability of the muscles[1] is generally found to disappear in from eight to ten months, developing simultaneously very marked amyotrophic changes.

It would be surprising if in other severe cases, or in cases of complete section, this muscular excitability of the faradic current should last twenty months without any nervous impulse being able to reach the muscle.

With this accumulation of contradictory facts, all we can conclude is that in a large number of cases *the*

[1] The maximum intensity of the galvanic current used was of 25–30 milliampères.

faradic contractility can appear in the muscles before voluntary motility, and this applies both to sections of the nerves which have been immediately sutured, and to physiological interruption of the nerve by a severe lesion.

It may be that in these cases faradic excitability has never at any time completely disappeared, and this one can easily understand, since there are certain kinds of paralysis existing with partial R. D., and that it has been masked by infiltration of the superficial layers which develops during the first few months after the wound, and which very probably also changes the structure of the muscle.

This infiltration can easily be detected in paralysis of the sciatic nerve. It is present, though less marked, in musculo-spiral paralyses.

The return of faradic excitability begins to appear in muscles which are atrophied, but still retain, or have recovered, a certain consistence and a certain degree of tonicity on deep palpation.

This faradic recovery takes place very slowly, but it progresses in a methodical manner right up to the time that voluntary movement appears, and occasionally it diminishes or even disappears for a short time when voluntary motility is about to return. On the contrary, in many other cases, especially in complete sections which have been sutured, or after resections, voluntary motility returns before electric contractility, and during the first few weeks, occasionally during the first months, which follow the onset of motor recovery, it is possible to find faradic inexcitability, or even galvanic inexcitability, of muscular masses which have been previously affected by paralysis and complete R. D.[1] The electric current is rapidly diffused into the normal muscles in the neighbourhood.

Clinical researches will still be necessary, with direct electrisation of the paralysed muscles exposed in the operation wound, in order to clear up these cases.

[1] Babinski has recorded a case of this kind, *Société de Neurologie*, May 6, 1915. *Revue Neurologique*, July 1915, p. 553.

In any case, whenever there is a serious lesion of the nerve which has not been operated on, or in case of section followed by suture with complete paralysis of the muscles and complete R. D., we continue to consider as a promising sign the appearance of faradic excitability in some of the paralysed muscles, no matter how slight this reaction may be.

This return generally coincides with signs of sensory recovery and an arrest in the advance of the amyotrophy. The subsequent history of a large number of cases has confirmed this.[1]

[1] Chinay, G. Bourgnignon, and Dagnan-Bouveret, while investigating the conditions of recovery of *galvanic* contractility, explain the early return of normal reactions in certain nerve injuries by the presence in the paralysed muscles of " a certain number of young regenerated fibres " in insufficient quantity for their contractions to determine a voluntary movement, but which, when examined electrically, may respond by a sharp contraction without inversion (*Paris Médical*, September 1916).

CHAPTER IV

RECOVERY OF VOLUNTARY MOTILITY

In studying the clinical forms of nerve lesions we have described the manner in which motor recovery takes place in each nerve-trunk of the limbs, and noted the order in which the muscles successively regain their functional activity; we need not, therefore, refer to this subject again.

Before the appearance of movements sufficiently marked to result in the displacement of a limb into which the terminal tendon of the muscle is inserted, one can convince oneself by *palpation* of the swelling of the muscle under the voluntary efforts of the patient.

Generally speaking, with severe lesions of the nerves, especially in cases of section or crushing of long standing followed by considerable atrophy of the muscles, *a certain degree of muscular tonicity returns before any reappearance of voluntary motility.*

This phenomenon is far less definite in those lesions in which atrophy has not led to marked emaciation of the muscles, but it can be made out even in those cases.

The appearance of this tonus does not necessarily involve an improvement in the defective attitude of the injured limb. Numerous causes can intervene to accentuate a paralytic position—*e.g.* articular ankylosis, laxity of the ligaments, and so forth.

Even when nervous impulses reach the muscle its movements remain at first awkward and uncertain,

and do not take place every time when the patient orders the limb to move, nor do the impulses reach all the muscles which are recovering.

For example, when a patient suffering from an injury to his musculo-spiral wishes to extend his wrist, his supinator longus contracts powerfully, and often in excess of the radial muscles.

A. Thomas attributes this to " an error in the switching " of the motor fibres which have not taken the right direction, and have not encountered each time the sheaths destined for their reception. Possibly it may also be due to a certain amount of inco-ordination and muscular weakness, or to forgetfulness of their previous function, and to the tendency to put into play too large a number of muscles when a fresh movement is to be executed.

Once the movement has been performed it is rapidly exhausted.

The patient does not, as a rule, acquire power, skill, and delicacy in isolated movements until after a long period of training.

We must remind the reader that in order to appreciate recovery of voluntary motility it is necessary to examine the case repeatedly and minutely, so as to detect the part played by auxiliary muscles, and to avoid being misled by the contraction of antagonistic muscles.

As Claude [1] has many times remarked, adduction movements of the thumb dependent on the ulnar nerve may simulate a movement of opposition. Adduction of the thumb, when the ulnar nerve is paralysed, may be initiated and simulated by the extensor longus pollicis.

The energetic contraction of the radial muscles causes hyperextension of the wrist, and determines a sort of " hollowing out of the hand " (Claude) which simulates a movement of flexion of the fingers.

In cases of simultaneous lesions of the median and ulnar nerves flexion of the wrist can be obtained by

[1] H. Claude, *Revue Neurologique*, April–May 1916, p. 493.

the combined action of the abductor longus and extensor brevis pollicis.

The anomalies of innervation of the muscles of the thenar eminence make the interpretation of their motor recovery rather difficult.

Froment [1] has suggested several signs which allow one to estimate the isolated action of each of these muscles.

Thus in order to judge of the return of functional activity in the flexor brevis pollicis, the classical test of opposition to the extremity of the little finger can still be used, or the patient may be asked to grasp a small glass.

Duchenne (of Boulogne) recommends the following test of the functional activity of the abductor brevis pollicis: opposition to the extremity of the index finger, first phalanx bent, last phalanges extended (hand in the shape of a duck's bill).

The condition of this muscle can still better be estimated (Froment) when a big cylinder or a bottle or a tumbler of large size is grasped.

During the regeneration of the nerve, and while voluntary motility is becoming established, another phenomenon to which André Thomas [2] has drawn attention may be seen, viz. muscular hypertonus, which is not uncommon in paralyses which are improving, and especially in certain cases of lesions of the musculo-spiral.

Together with this phenomenon, which may be localised in some of the paralysed muscles, one may observe the presence of cutaneous sensations in the territory of the musculo-spiral nerve, which can be elicited by pressure on certain muscles, such as the extensor communis or the supinator longus.

[1] J. Froment, *Revue Neurologique*, April–May 1916, p. 508.

[2] A. Thomas, "Hypertonie musculaire dans la paralysie radiale en voie d'amélioration. Sensations cutanées dans le domaine du nerf radial, provoquées par la pression de muscles qui reçoivent leur innervation du même nerf. Égarement des cylindraxes régénérés, destinés à la peau dans les nerfs musculaires," *Société de Neurologie*, July 29, 1915. *Revue Neurologique*, August–September 1915.

Thomas explains this fact by the diversion of the sensory axis-cylinders which were destined for the cutaneous area into sheaths belonging to muscular fibres.

The presence of sensory axis-cylinders in contact with muscular fibres would then give rise to cutaneous sensations, and possibly also to an irritation of the muscle, tending towards contracture.

CHAPTER V

SIGNS OF SEVERE LESIONS OF THE NERVES

THE main object of the physician when faced with a nerve wound is to try to appreciate the severity of the lesion from the very start in order to know what treatment to adopt.

If it were possible to be certain at once that there was complete section, immediate suture of the nerve could be performed, as an early operation is the best condition for its success; but this certitude can only be acquired after observing the wound for several months, and after having noted the absence of any sign of recovery.

Neurologists and surgeons have made numerous attempts to discover any signs which would justify a more rapid diagnosis and furnish definite indications for operation from the very start.

These researches, we must admit, have not yet resulted in any definite conclusions.

The most important and complete work on this subject has been that of Mme. Dejerine and J. Mouzon. In the course of a series of articles these authors have described the signs which they consider as justifying the diagnosis of an anatomical nerve lesion.[1] They have distinguished several symptom-complexes, which they have termed *syndromes of interruption*, *syndromes*

[1] M. and Mme. Dejerine and M. J. Mouzon, "Les lésions des gros troncs nerveux des membres par projectiles de guerre. Les différentes syndrômes eliniques et les indications opératoires," *Presse médicale*, May 10, July 8, and August 30, 1915.

of compression, syndromes of irritation, syndromes of dissociation, and *syndromes of recovery.*

A. **The Syndrome of Interruption** justifies the diagnosis of *complete histological interruption* which is present whenever " the lesion prevents any axis-cylinder of the upper segment from passing into the lower segment, *whatever the macroscopic* or *naked-eye appearance may be.*" Once we have admitted the lesion to be of such a degree of severity, surgical interference is indicated. Even if it is found on operation that the nerve is not divided, but merely presents a swelling or a " nerve cheloid," M. Dejerine, Mme. Dejerine, and Mouzon advise resection of the nerve and suture. All that they require is that the syndrome of interruption should first have been found by clinical examination.

Let us first of all study the symptoms constituting this clinical syndrome; we will then mention the criticisms that have been made :—

1. *Muscular Signs.* —Three fundamental signs exist :—

(1) *Complete paralysis* of all muscles supplied by the nerve below the lesion.

(2) *Complete absence of tonicity*, evidenced by softness and flaccidity of the muscles on palpation, and by the position of the limb in repose ; wrist-drop (musculospiral), very marked ape-hand (median), irreducible claw-hand (ulnar), excessive varus equinus (external popliteal), and foot-drop (great sciatic).

(3) *Absence of any pain on pressure of the muscles supplied by the injured nerve.* This muscular analgesia is stated to persist some time even after the regeneration of the nerve and the reappearance of tonicity and voluntary motility.

Further *muscular symptoms* of *secondary* importance are :—

(*a*) Abolition of corresponding reflexes (tendinous, periosteal, and cutaneous).

(*b*) Exaggerated excitability of the muscle to mechanical stimulus.

(*c*) Amyotrophy.

(*d*) Certain deformities, such as dorsal swelling of the carpus or tarsus.

(*e*) Complete reaction of degeneration in the paralysed muscles.

2. *Sensory Signs.*—These are characterised by :—

(a) *Absence of any zone of hyperæsthesia or paræsthesia in the region supplied by the injured nerve.*

(b) *Absence of pain on pressure applied to the nerve-trunk below the lesion.*

(c) *Localisation of the changes in objective sensibility as well as their topographic distribution,* which embraces the whole cutaneous, osseous, and articular region supplied by fibres of the injured nerve which have been given off below the seat of the lesion.

B. **Syndrome of Irritation.**—This comprises painful injuries of nerves, on which we have sufficiently dwelt in our first volume.

C. **Syndromes of Dissociation** relate to the numerous partial lesions of the nerves, and give rise to incomplete paralysis.

D. **The Syndrome of Compression**, which indicates lesions surrounding the nerve rather than in its actual substance, is characterised by the same phenomena as those described in the syndrome of interruption, but in a less degree. What distinguishes this condition from the last, according to observers, is the presence of pain when the muscles and the nerve-trunks are subjected to pressure and the lesser degree of impairment of objective sensibility which generally consists in hypæsthesia.

E. Finally, the **Syndrome of Regeneration** of the nerve, " which may follow any of the previous syndromes," comprises :—

1. *Sensory symptoms*, viz. *spontaneous pain* radiating throughout the whole course of the affected nerve ; *pain on pressure of the nerve-trunks* ; *shrinkage of the anæsthetic areas and appearance of paræsthesia* (protopathic sensibility of Head).

2. *Muscular symptoms* subsequent to the signs of

sensory recovery, mainly characterised by the *return of muscular tonus* (gradual modification in the position of the limb).

Such in outline is the very interesting and elaborate theory worked out by M. and Mme. Dejerine and Mouzon.

We think that the conditions met with in actual practice rarely assume this schematic appearance, and that the clinical varieties which the doctor has to examine do not always correspond to this description.

The only syndrome which we shall discuss is that of *complete interruption*, on account of its great practical importance. If this syndrome was allowed absolute diagnostic value each time that it was encountered, operation would be necessary, and even if the operation did not show actual section of the nerve, resection followed by suture would nevertheless be indicated.

In accordance with Prof. Pitres, we believe that it is impossible to make a definite diagnosis of anatomical or histological interruption of the nerve, but only one of *physiological interruption*. "For even if loss of conduction necessarily follows transverse section of the nerve, it can just as well be the result of injuries which have affected the nerve-fibres without dividing the neurilemma—such as contusions, compressions, constrictions, and possibly even mere commotion of the nerve-trunk."[1]

Resection among other dangers would then result in interrupting for a long time a regeneration which was tending to take place spontaneously.

The practical side of the question has been well brought out by Henry Meige. "A scientific distinction between anatomical section and physiological section or interruption is a thoroughly judicious

[1] A. Pitres, "La valeur des signes cliniques permettant de reconnaître dans les blessures des nerfs périphériques: *A*. La section complète du nerf; *B*. La restauration fonctionnelle," *Société de Neurologie*, April 6, 1916. *Revue Neurologique*, April–May 1916, p. 477.

one, but I fear that in practice it is not always very carefully interpreted. Is not one liable occasionally to confound impairment in the function with mutilation of the organ? And is there not a possibility of recommending the same operation for nerves which have undergone physiological interruption as for those with anatomical section? I foresee a very real danger to which attention should be drawn.

"Circumspection is all the more necessary since the signs by which we diagnose a section or physiological interruption are still under discussion—their symptomatological value varies according to different observers—and moreover these signs are only temporary, and in fact do on occasion become modified or disappear spontaneously without intervention.

"Hence we must ask ourselves whether the presence of these signs is of sufficient value to justify the resection of a nerve-trunk which is not completely severed."[1]

The originators of the syndrome of interruption expressly state that value can only be attached to the syndrome when all its constituent signs are present at the same time.

Any one of these signs taken separately is only of doubtful value.

Thus *complete paralysis* of all the muscles supplied by the nerve below the seat of the lesion is an absolutely essential sign for the diagnosis of a complete interruption of the nerve. No one would think of maintaining such a diagnosis if a few of the muscles supplied by the nerve preserved their motility. Nevertheless there are some nerves—and these are the very ones most frequently injured, such as the musculo-spiral or external popliteal—which react to injury in the majority of cases by a generalised and immediate paralysis of all the muscles without the lesion being necessarily very serious.

With regard to the *absence of tonicity of the muscles*,

[1] Henry Meige, *Société de Neurologie de Paris*, April 6, 1916. Cf. *Revue Neurologique*, April–May 1916, p. 499 and following.

it should be recognised that flaccidity of the muscle on palpation varies in each subject.

"Muscular tonicity varies considerably in the normal state. Individuals may be constitutionally hyper- or hypotonic. Some have short, bulky, and hard muscles which never relax completely; while others have long, thin, soft muscles in which relaxation is excessive. The same condition can be observed in the tendons, ligaments, and aponeuroses. . . .

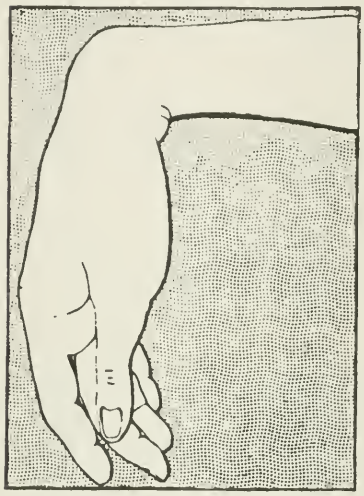


Fig. 18.—Musculo-spiral paralysis in course of recovery. Great hypotonus of extensor muscles of wrist, despite fairly good motor and electrical recovery in these muscles.

"We have seen muscles in which hypotonus and flaccidity were excessive recover their contractility without operation. The flaccidity of the muscles may be masked by concomitant lesions in the muscles, tendons, or aponeuroses, or by an œdematous infiltration, which is not uncommon, especially in the lower limbs. We have seen several cases where the flaccidity and atrophy of the muscles only became apparent after the subsidence of long-standing œdema; at the same time appeared definite signs of sensory and electrical and then motor recovery" (Henry Meige).[1]

The *attitude of the limb at rest* is of still more debatable diagnostic value. *Hypotonus* may be very marked in the course of reflex paralyses and functional pareses (Babinski). It is sometimes very pronounced in severe injuries to nerves, which nevertheless recover in a few months. We have often seen musculo-spiral paralyses with marked wrist-drop

[1] Henry Meige, *loc. cit.*

(fig. 18) or paralyses of the external popliteal accompanied by excessive equino-varus (due to ligamentous distension) recover, both from the motor and electrical point of view, without either the attitude of the hand or foot altering in any appreciable manner.

What we have said of the variability of *ulnar claw-hand*[1] allows us to state that the existence of a claw-hand depends upon too many factors for one to be able to regard it as a definite diagnostic sign.

The interpretation of the "ape-hand" seen in lesions of the median may be complicated by an anomalous nerve-supply.

The *absence of any pain on pressure of the muscles* is a sign which Henry Meige and Pitres have discredited. It is indeed a very uncertain phenomenon. The muscles often present the same painlessness on deep pressure as on the sound side. In other cases, as many observers have recorded, in which pressure on the muscles was painful, a subsequent operation has shown that the nerve was completely divided with both ends apart. This has occurred fairly frequently in the musculo-spiral and external popliteal. In many cases this pain, which is sometimes very intense, is due to concomitant injuries to adjacent bones, tendons, or aponeuroses, or to the involvement of sensory filaments belonging to other nerves which supply the skin over the paralysed muscles.

We shall not criticise the motor and sensory signs, which the authors themselves declare to be but of secondary importance and which we shall discuss later.

From the practical point of view, we will remind the reader that a large number of cases of complete paralysis showing all the severe signs described under the name of "syndrome of interruption" recovered without any operation, or after simply freeing and clearing the nerve. In other cases considered to be of a very severe nature, and of several months' duration, in which operation had shown a large, hard

[1] Mme. Athanassio-Benisty, *Clinical Forms of Nerve Lesions.*

fibrous swelling, with or without a lateral notch, the pseudo-neuroma was excised and end-to-end suture performed. Histological examination of these excised neuromata conducted by Pierre Marie and Foix showed that very often some of the axis-cylinders of the upper end had systematically invaded the cicatrix, and were advancing, for the most part, towards the lower end, which some of them had even reached. Regeneration was, in fact, taking place spontaneously.

Is there then no sign, no symptom or combination of symptoms, which will allow one to state with certainty that a nerve has suffered anatomical section —a diagnosis which is of the utmost importance as regards surgical treatment?

We regret that we are forced to reply in the negative and to repeat what Prof. Pitres says: "It is possible to diagnose physiological interruption of a nerve, but it is impossible to diagnose its anatomical section."

Physiological interruption is capable of spontaneous repair in a large number of cases. It is not, therefore, justifiable to recommend resection of a nerve as a routine treatment.

It is necessary to wait several months before one can state with any certainty that a physiological interruption of a nerve is severe and that it corresponds to an histological interruption. During this period it is essential to make repeated and careful examinations; and then, if there is no sign of regeneration, and if the original symptoms suggestive of a severe lesion have not undergone any change, and not till then, should an attempt be made to induce by resection followed by suture a regeneration which it appears cannot take place spontaneously.

What then are the signs of a severe lesion which should be noted from the first?

Signs of a Severe Lesion

In our opinion these signs are as follows:—

1. **Total Paralysis of all the Muscles below the Lesion.** —This symptom is of most value in wounds of the

median and ulnar, as these nerves so often show only incomplete or dissociated paralyses as the result of injuries received in warfare.

It is of less value in the lesions of the musculo-spiral and external popliteal nerves.

2. **Complete Reaction of Degeneration,** which can be found from an early date (six or seven weeks after injury), and in which the quantitative and qualitative alterations *become more marked at each subsequent examination.* In the case of a severe lesion the faradic excitability of the muscles rapidly disappears, galvanic hypoexcitability becomes pronounced, and the sluggishness of the contraction increases, being very frequently accompanied by polar reversal and displacement of the motor points, resulting eventually in the longitudinal reaction which finally predominates over excitability at the motor points and persists alone several months afterwards. At a certain moment, as we have said, electrical excitability both to the continuous and interrupted current disappears absolutely. This *progressive disappearance* is a grave sign.

3. **Rapid and Extensive Atrophy of the Paralysed Muscles** is easily detected in the posterior part of the forearm in the case of the musculo-spiral (with wrist-drop); in the epitrochlear muscles, in contrast to the prominence of the supinator longus, in the case of the median; in the whole leg in the case of the sciatic; and in the antero-external part of the leg in the case of the external popliteal (with marked equino-varus).

Atrophy of the interossei and of the hypothenar eminence in the case of the *ulnar* nerve does not possess the same importance, since it occurs even when the lesion is not very severe.

4. **The Presence of Certain Thermal and Vasomotor Disturbances,** in addition to the preceding signs, is of great assistance in diagnosis. They are as follows:—

Considerable *lowering of the local temperature* (in the absence of any vascular lesion) of the skin covering

the atrophied muscles. This refrigeration is still more noticeable when the skin is supplied by the injured nerve.

Fig. 19.—Distribution of sensory changes in severe lesions of the great sciatic.

Black: complete anæsthesia for all stimuli. *Grey*: hypæsthesia.

Palpation reveals this sign: on the posterior surface of the forearm in the case of musculo-spiral paralysis; on the hypothenar eminence and over the little finger in the case of the ulnar; in the index finger in the case of the median; on the antero-external surface of the leg in the case of the external popliteal; and over the whole calf in the case of the great sciatic. In the last case, if the foot is *warm and dry* and slightly succulent, this is another sign of gravity. Frequently there is also a slight œdematous infiltration of the malleolar region and even of the calf.

5. **Absence of Pain when the Trunk of the Nerve is subjected to Pressure below the Seat of the Lesion.**[1]—Nevertheless in the course of complete division of the musculo-spiral and in certain lesions of the great sciatic, or of the external popliteal, there may be painful sensibility on pressure of the nerve-trunk below the seat of the lesion.[2]

[1] The following are the points of election for palpation of each nerve:—

Musculo-spiral.—The groove of the humerus, neck of the radius, and external border of the forearm.

Median.—Inner aspect of arm, course of the nerve in the forearm (middle of anterior aspect), and thenar eminence.

Ulnar.—Groove between internal condyle and olecranon, and course of the nerve in the forearm (inner border).

Great Sciatic.—Course of the internal popliteal (middle of the popliteal space), posterior tibial (middle of the calf), and external popliteal (neck of the fibula).

[2] Claude has also noticed cases of this type, especially in the course of a complete section of the musculo-spiral. *Revue Neurologique*, April–May 1916, p. 493.

6. **Considerable Disturbances of Objective Sensibility.**—In lesions of the *great sciatic* these disturbances, in order to have any value, must affect the whole foot, except its internal border and the internal malleolus. The loss of deep sensibility on pressure and pinching is absolute, as is also the abolition of vibratory sensibility in the bones and the complete

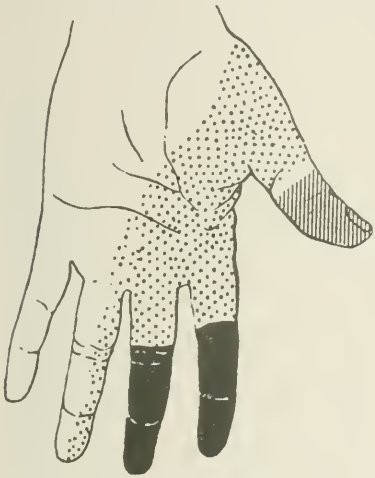


FIG. 20.—Palm of the hand.

FIG. 21.—Back of the hand.

Distribution of sensory changes in the course of severe lesions of the median.

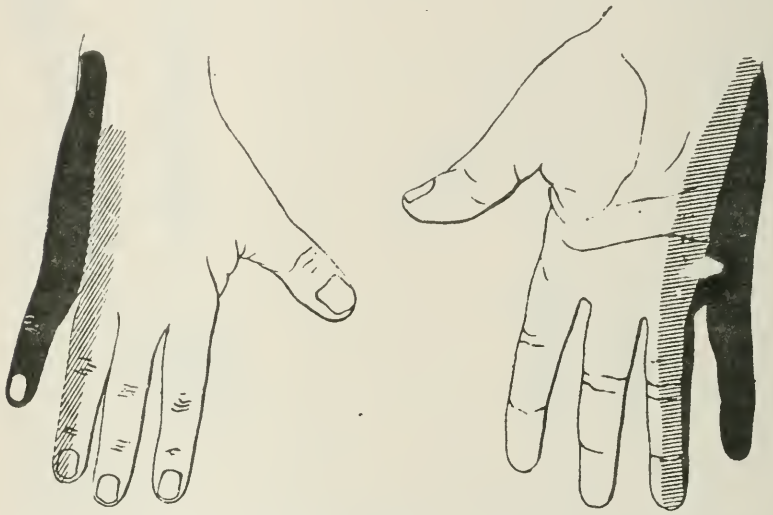
Black: complete anæsthesia for all kinds of stimulation. *Grey*: hypæsthesia to pricking, anæsthesia to heat and cold. *Dotted area*: less marked hypæsthesia.

absence of the sense of position in the toes or even the instep. In the leg this anæsthesia extends to the antero-external surface of that segment and comes more or less close to the knee (fig. 19).

In the case of the median, anæsthesia is complete in the last two phalanges of the index and middle finger, with total loss of deep sensibility and the sense of position. If the anæsthesia extends to the thumb and the thenar eminence, this sign is of still greater importance (figs. 20 and 21).

In the case of the *ulnar*, sensory disturbances are very frequent and marked. The most significant are the complete loss of sensibility of all kinds in the little finger and on the inner part of the hypothenar eminence (fig. 22).

In the case of lesions of the *external popliteal*, the anæsthesia occupies the middle region of the dorsal aspect of the foot and a narrow tract running up the



FIGS. 22 and 23.—Area showing sensory changes in severe lesions of the ulnar.

Black: complete anæsthesia for all kinds of stimulation. *Grey*: hypæsthesia to pricking, anæsthesia to cold and heat.

outer surface of the leg as high as the junction of the middle and upper third (fig. 24).

This typical distribution may not occur, and we have observed cases of complete anatomical section of the external popliteal in the popliteal space in which the anæsthetic area was confined to the zone of the anterior tibial. The supplementary nerve-supply was probably due in this case to the external saphenous.

We have seen how inconspicuous and inconstant

were the sensory symptoms in the course of lesions of the musculo-spiral. Nevertheless a patch of anæsthesia situated over the first dorsal interosseous space possesses some value.

Such are the main signs which claim the practitioner's attention.

Other less important symptoms must be mentioned. Their existence may form a further argument in favour of a severe lesion or complete division of the nerve. Their absence, on the other hand, is of far less significance.

They are as follows :—

Loss of the osteo-periosteal tendon and cutaneous reflexes. — We have already alluded to their variability.

Exaggeration of the Mechanical Excitability of the Muscle.—This is far from giving any satisfactory information as to the nature of the lesion. *Slowness of idio-muscular contraction* is a sign of the same import as the sluggishness of the contraction produced by the continuous current, but it is not so constant.

Vasomotor and trophic disturbances are always present in severe lesions of the nerve, but one must always remember the possibility of the presence of a vascular lesion, and bear in mind the character of the reactions peculiar to certain nerves such as the median or the great sciatic.

The *abolition of the secretion of sweat* is of value when present, and is almost always found in severe injuries to the median, ulnar, and great sciatic.

FIG. 24.—The zone of sensory changes in lesions of the external popliteal nerve.

Black : anæsthesia. *Grey*: hypæsthesia.

The researches of Claude and Chauvet [1] and of René Porak [2] have shown that the disappearance of sweating

[1] Claude and Chauvet, *Sémiologie réelle des sections totales des nerfs mixtes périphériques.* Paris, published by Maloine, 1901.

[2] R. Porak, *Société Médicale des Hôpitaux*, July 30, 1915.

is observed and localised in the region of a nerve which has undergone complete anatomical division.

Certain *trophic changes* must also be considered, although too much value must not be given them. They are as follows :—

The presence of *ulceration* without any vascular lesion being present (Léri).

Hypotrichosis, or the lessening in number and size of the hairs in the zone supplied by an injured nerve. Maurice Villaret [1] has drawn particular attention to this sign. Hypertrichosis, on the other hand, would rather suggest an irritation of the nerve.

The same may be said of changes in the *finger-prints* recently studied by Cestan, Paul Descomps, and Euzière.[2] In cases of severe injury to the median and ulnar nerves a breaking-up and mazing of the outlines may be seen, and is said to have the same significance as real trophic disturbances.

André Thomas has drawn attention to a series of signs from his study of the *cutaneous cicatrix* and the region around this cicatrix.

Stimulation of this zone by a fine brush or a needle gives rise to sensations which are localised to the area stimulated and are also transmitted to the periphery of the nerve (synæsthesia).

" This *cicatricial topo-parœsthesia* shows that there is at any rate a partial division of the nerve, and that this division is absolute ; this is the most important point which must be insisted upon in the presence of a syndrome of complete paralysis " (André Thomas).

In addition to this sign, one can usually find by palpation a swelling in the course of the injured nerve.

Among all these signs which we have just described,

[1] Maurice Villaret, " Contribution à l'étude des troubles du système pileux et de la sudation spontanée des membres au cours des lésions traumatiques de guerre des nerfs périphériques," *Société Médicale des Hôpitaux*, December 17, 1915, and *Revue Neurologique*, April–May 1916, p. 495.

[2] Cestan, Paul Descomps, and J. Euzière, " Les altérations des empreintes digitales dans les lésions des nerfs périphériques du membre supérieur," *Société Médicale des Hôpitaux*, May 5, 1916.

we would attach most importance to the *rapid and progressive atrophy of the muscles* and to the *progress of their electrical degeneration.*

The presence of this sign, six to eight weeks after injury (the time at which the patients usually reach the neurological centres) justifies the diagnosis of total and complete paralysis.

The subsequent course, accompanied by careful and repeated examinations showing the persistence or aggravation of these signs, indicates a severe injury.

The disappearance of these symptoms, on the contrary, will lessen the probability of such an injury.

" In general, one should be guided less by an association of clinical signs than by the *evolution* of these signs. Thus the diagnosis of complete physiological interruption of a nerve cannot be made from a single examination, however carefully conducted. Even if this examination enables one to find all the principal and secondary signs of a complete interruption, it must be followed by many other examinations at sufficiently long intervals to enable one to detect any progressive aggravation, or obstinate stagnation which could justify an exploratory operation" (Henri Meige).

The period necessary for the regeneration of the nerve is therefore variable. It depends mainly on the anatomical condition of the nerve, though the factors which are favourable or otherwise to its regeneration are unknown to us, and also on the nature of each nerve, the clinical individuality of the nerves being shown by the rapidity and ease with which they regenerate.

The first signs of sensory recovery, if they occur, may appear alone for a long time without being followed by a progressive and regular regeneration of the other functions of the nerve.

Under these conditions how long must we wait before being able to form a strong presumption in favour of a severe anatomical lesion, and before advising an operation ?

Is there no danger, in waiting too long, of degeneration occurring in the nerve-trunk, the muscles and the

terminal sensory and motor organs, thus rendering all regeneration impossible in future ?

From the cases which we have observed, and which we shall describe later, we think we are justified in stating that there is no danger in waiting four or five months after the infliction of the wound.

On the other hand, if no sign of regeneration appears, it is wise to operate towards the fifth month, since further delay may impair the ultimate success of the suture.

When the severe signs which we have detailed persist or increase towards the seventh or eighth month, one can assume a *complete section with or without pseudo-continuity of the nerve.* All the more so is this the case when one sees the wounded for the first time, nine, ten, or twelve months after the injury.

One may ask if when in doubt it would not be better to operate as soon as paralysis is considered to be complete, and to resect and suture every injured nerve, since we cannot know from the start the gravity of the nervous cicatrix caused by the projectile. Is it not logical to replace an irregular inflammatory cicatrix by a regular aseptic one suitable for the passage of the axis-cylinders to the peripheral end of the nerve ?

This question can best be answered as follows : a large number of sutures with freshening of the two ends of the nerves performed within six weeks to four months after the injury (in other words, fairly soon), and in which healing has occurred by first intention, have resulted in failures, or else progress has ceased at an early stage of regeneration.

The same applies to many cases of total or partial resection followed by sutures performed under favourable conditions, and according to the most logical methods.

These facts are particularly true in the case of lesions of the median and great sciatic nerve.

If nerve suture, which should always be attempted in the case of complete division of the nerve with separation of both ends, is not always successful, have

we the right to resect a nerve whose anatomical continuity is intact and in which naked-eye examination does not show any severe crushing nor any appearance equivalent to complete section such as we have described previously ?

We do not think that there is any justification for such procedure.

Order in which the Signs of Regeneration Appear

Which are the earliest symptoms indicating the regeneration of a nerve, and what is their value ?

As we have already described them, we will now see in what order they appear :—

1. *Signs of sensory regeneration appear first.* The earliest in the order of their appearance are *pain when the skin is pinched* in the sensory area of the nerve, *pain when the nerve is pressed* below the lesion, *formication* produced by this pressure along the nerve below the probable seat of its injury, and *spontaneous* and *provoked aching* in certain muscles. This sign is particularly marked in wounds of the sciatic.

These symptoms, as we have said, are early ; they may develop in certain injuries of the nerves after three or four months, sometimes earlier, and occasionally later.

They precede by a long time the other signs of sensory regeneration, and also of motor and electrical recovery.

Occasionally nervous regeneration seems to halt at this first phase. We have seen a few cases which have not been operated on and more numerous ones in which the nerve has been operated on under perfect conditions and sutured end to end, and others in which the nerve has simply been freed and in which after twelve to eighteen months nervous regeneration had stopped at this stage of *initial sensory recovery.*

2. The *muscular signs*, which should be looked for carefully and at frequent intervals, are the next to

appear. The *arrest of the atrophy*, which does not result in the complete emaciation only produced by total and definite section, is of great importance.

A *certain consistency* and tonicity of the muscles on palpation, and, more important still, the reappearance of this consistency after a period of progressive atrophy, is a sign of good augury. This sign is difficult to detect in certain cases of wounds of the sciatic with infiltration of the skin.

The change in the attitude of the limb is a sign of less value. The foot-drop may persist in wounds of the great sciatic even when motor regeneration starts in some of the muscles, and a certain degree of tonus can be found when the muscles are palpated. The same thing applies to wrist-drop in wounds of the musculo spiral.

The relaxed condition of the ligaments, articular lesions, and occasionally vasomotor disturbances often prevent correct appreciation of the change in the position of the limb which *a priori* should always result from the increase in muscular tonicity.

Nevertheless an improvement in the *dangling attitude of the toes* in some lesions of the sciatic, the modifications in the course of the ulnar claw-hand coincident with other signs of regeneration, are of good import and should claim our attention.

3. In some of the muscles which have preserved or recovered some of their tonicity, one can find, four or six months after injury, sometimes later, a slight degree of faradic contractility.

The contraction may be quite trifling, occasionally quite localised, sometimes only perceptible on palpation, or, on the other hand, sufficiently marked to occasion a distinct elevation of the tendon of the muscle that is being stimulated.

As we have already described the various forms of electrical recovery, we need not return to this subject. We will simply mention that the persistence of faradic excitability, its progressive increase, and its spread to other paralysed muscles are of favourable prognostic

import, and allow us to hope for a return of voluntary motility in the near future. The muscle which has first recovered its faradic contractility will usually be the first to regain its motility.

Motor recovery is less certain in cases in which excitability by the induced current varies from day to day, and does not progress in a regular manner.

The *disturbance of objective sensibility* subsides and gradually goes through those stages which we have described previously, while the sensory signs of the early stage persist (formication and pain on pressure of the nerve and skin).

Muscular atrophy being arrested, the firmness of the muscles increases, and finally *voluntary movements* begin.

Muscular contraction is at first shown by a simple lifting of the muscle in the course of certain contractions of antagonistic muscles, but it becomes stronger and finally reaches the tendon. The movement is uncertain and weak at first, but gradually improves.

How long does it take for regeneration of the nerve to occur, by which we mean the first sign of motor return in a part or the whole of the paralysed muscle supplied by the injured nerve?

Nothing is more variable. The time required is never less than five months in slight cases, and it may be as much as eight or ten months in severe cases and after early nerve suture of certain nerves such as the musculo-spiral or external popliteal; it may reach twelve, fourteen, or sixteen months in some very severe instances, and in late sutures of the nerves mentioned above, or in some early sutures of the ulnar, median, or great sciatic.

Possibly we may see eventual nerve regeneration after a longer period still even in those which we now consider lost, despite sutures, grafts, and freeings. There is nothing to prevent us hoping for appreciable improvement in certain severe and complete paralyses which have not been operated on, and in which we see signs of commencing sensory recovery.

CHAPTER VI

SURGICAL TREATMENT OF INJURIES TO NERVES

From the beginning of the war both neurologists and surgeons were surprised by the high incidence of wounds of the nerves (20 per cent. of all wounds), and anxiously sought for the best treatment to apply to these cases.

At first both neurologists and surgeons agreed to avoid the use of the knife, since the first operations did not give those results which pre-war neurological and surgical literature had led them to expect. These operations neither relieved pain nor hastened motor recovery.

The operative technique was found fault with, or the blame was laid on the suppuration of the tissues, or the peculiar nature of wounds of the nerves in war, which always tear the tissues extensively and are accompanied by excessive fibrous reaction.

It must also be confessed that the knowledge of nerve degeneration and regeneration had been somewhat forgotten.

There was therefore a period of indecision, after which the neurologists adopted the surgical principle of exploration, and henceforth for a time handed over most of their cases to the surgeon.

Both parties acted logically and wisely in allowing themselves to be guided by the data furnished by clinical examination and the anatomical appearance of the lesions. Several essential rules were decided upon,

e.g. the importance of not interfering before complete cessation of all suppuration, the advantage of cleaning up the area of the lesion, and the need of protecting the nerve against any return of suppuration followed by sclerosis.

Another important point was also established, viz. the delicate nature of nervous tissue and the necessity of taking the greatest care in manipulating it. The logical sequence of this was the axiom : *a nerve-trunk in which there is no solution of continuity should not be divided or resected.*

There were certainly a few daring attempts to resect exuberant nerve cicatrices in order to suture the nerve in healthy tissue and thus to ensure a more rapid and theoretically more certain regeneration. Delorme in particular proceeded in this manner with regard to lesions of the great sciatic. The Surgical Society criticised this method, and we do not know what was the subsequent history of cases thus operated on.

The fact remains that a large number of surgeons soon discarded any operations on nerves, discouraged both by the slowness of nerve repair in general and sutures in particular. A few of them even went so far as to declare every nerve suture to be a mistake and destined to failure from the start.

On the other hand, the campaign conducted by M. and Mme. Dejerine, and their definite advice to resect every cicatrix of a nerve-trunk manifested clinically by the " syndrome of complete interruption," without any sign of regeneration three weeks after receipt of the wound, influenced other surgeons, who resumed resecting. There are as yet no definite statistics which would enable their results to be gauged.

We consider it advisable to report those cases which we have been able to follow up personally, and to compare them with other cases in the various neurological and surgical centres.

During the last months of 1914, and at the beginning of 1915, Professor Pierre Marie had a large number of cases under his care at La Salpêtrière suffering from

nerve wounds, operated on by Gosset and Pascalis for severe or painful lesions, and less severe lesions with incomplete paralyses and some evidence of regeneration. It was to be hoped that freeing and cleaning up of the nerve-trunk would assist in the recovery of its functions. On the other hand, it seemed indicated to suture the nerve in the case of division.

In some of the cases in which clinical examination showed the presence of a severe lesion the nerve was found to be crushed, or had a very hard and exuberant swelling; recourse was then had to resection followed by end-to-end suture.

In dealing with an anatomical section of the nerve, Gosset freshened the two ends and performed end-to-end suture.

Only in a few cases in which the distance separating the two segments was too great did he use nerve-grafting, the graft being usually taken from the cutaneous branch of the musculo-cutaneous, one of the two terminal branches of the external popliteal.

When the continuity of the nerve was unimpaired, the nerve cicatrix being represented by a "pseudo-neuroma of attrition," of which we have spoken before, without any crushing of the nerve or solution of continuity, Gosset, in accordance with Professor Pierre Marie, only freed the nerve or subjected it to "hersage," *i.e.* made a few slight longitudinal incisions over the cicatricial swelling, each 2 to 3 centimetres long, according to the requirements of each case.

Lateral notches were freshened and sutured.

Neuromata situated within the nerve-trunk were enucleated whenever possible.

The general results of these operations were as follows:—

First, and this is of the utmost importance, no aggravation of any kind was recorded. Our statistics comprise about 150 cases operated on.[1] We are able to state definitely that an exploratory incision in nerve wounds is quite harmless when the surgeon is

[1] About one-fifth of these cases were operated on by Pascalis.

sure of his technique, and conducts the operation with prudence and dispatch, and ensures absolute asepsis.

These operations, except in very rare instances, were performed after suppuration had completely subsided. The post-operative history was normal, and cicatrisation took place by first intention.

Among more than fifty simple *freeings* we have to record twenty complete cures; one failure (shrapnel fragment imbedded in the great sciatic in the region of the buttock; no voluntary movement had reappeared one year after operation). All the other cases were followed by improvement which was more or less rapid, but always progressive.

Hersage was performed when the anatomical lesion appeared of a more severe nature.

Out of about fifty to sixty, twenty-five were completely cured. One failure was recorded (great sciatic nerve in the region of the buttock), and there was undoubted and sustained improvement in thirty cases.

Among eleven *sutures*, we had two complete recoveries (both musculo-spiral), three partial and progressive recoveries (two ulnar and one external popliteal). The six other cases were distributed as follows: three musculo-spiral (sutured one year after receipt of wound), one ulnar, two median, one great sciatic. Nearly all these cases nevertheless show commencing sensory recovery, except the two median.

We were able to follow up five complete resections followed by sutures, of which two were almost completely cured (both external popliteal), one showed signs of commencing recovery (musculo-spiral), and two resulted in failure (one musculo-spiral, one ulnar).

Four *partial resections* were followed by two very satisfactory recoveries (one musculo-spiral, one external popliteal) and two failures (one median and one great sciatic).

Two musculo-spiral paralyse swith section and wide separation of the two ends of the nerve were treated by suture by means of a *nerve graft*. In these cases the nerves had been wounded eight to twelve months

previously. Twenty months after the operation we can find only vague signs of sensory recovery.

From these statistics several general conclusions can be drawn which have been confirmed by the work of other neurological centres :—

1. *Exploratory incisions* carefully performed under aseptic conditions are not harmful to the injured nerves.

2. *Freeings* do no harm and may favour recovery.

3. *Hersage* of neuromata do no harm.

It is a question, however, whether operations of this kind are essential. Thus, an injured nerve which has not suffered severe cicatricial damage and has maintained its continuity will probably be able to regenerate at the end of eight to twelve months without an operation of any kind.

4. *Sutures* for complete sections of nerves, and performed six to eight months after receipt of the wound, often result in cure in the case of the musculo-spiral and external popliteal.

5. *Resection* followed by suture in severe lesions of the same nerves gives equally good results when the operation has not been postponed for too long a period (eight months at the very utmost after the wound).

6. *Sutures or resections followed by sutures of the ulnar nerve regenerate far less quickly, and are altogether less satisfactory ; those of the great sciatic and of the median also regenerate extremely slowly ;* frequently twelve to eighteen months after operation there is no return of nervous function, or only a slight degree of recovery.

Such is the list of the results of operation which have been observed in Professor Pierre Marie's service at La Salpêtrière.

Comprehensive statistics are practically non-existent. Isolated reports and communications to learned societies mention some cases of nerve suture followed by motor recovery. Generally they refer to section of the musculo-spiral nerve with early suture

(two to six months after injury), but J. Heitz, in a report to the Health Service (May 1916), mentions a case of suture of the musculo-spiral performed very late, more than one year after receipt of the wound, and followed a few months after the operation by motor recovery.

We have also seen a case of this sort in which the musculo-spiral had been sutured one year after injury and where the power of voluntary movement began to return ten months after suture. Cases of this kind are of considerable interest and should be remembered.

Researches should be conducted along these lines in order to establish if it is advisable to interfere in old-standing cases of paralysis due to a wound, in which for some reason no operation has been performed.

Successes have less frequently been noted after suture of the ulnar, median, or great sciatic. On the other hand regeneration of the nerve is not uncommon in suture of the external popliteal.

René Dumas brought before the Surgical Society some careful statistics[1] in which he had collected hundreds of cases which had been operated on, and he was able to draw some interesting conclusions from his own experience.

René Dumas has had the opportunity of performing a considerable number of *sutures* and a very great number of *freeings*. He found that the recovery of movement did not occur until very late, but the point which struck him most was that there was a *definite individuality* in the nerves from the point of view of the rapidity and completeness with which they regained their motor power.

The musculo-spiral regenerates remarkably readily, and the ulnar only slowly, while the great sciatic (especially the internal popliteal) and the median give the most disappointing results.

Henry Meige has also pointed out that the musculo-

[1] René Dumas, "Libération des nerfs et récupération fonctionnelle," *Société de Chirurgie*, meeting held February 2, 1916. *Bulletins et Mémoires de la Société de Chirurgie de Paris*, February 8, 1916.

spiral is at once the most delicate of all nerves and the one which regenerates most satisfactorily both spontaneously or after surgical interference.

Tinel has compiled statistics of 108 cases of suture or of nerve grafts.[1] He casts no doubt on the success of nerve suture, and in 108 cases he had barely 12 to 15 per cent. of failures. Twenty-two cases were almost completely cured, and all the others are on the high-road to more or less rapid recovery; but at present we have only a brief summary of the clinical and anatomical features of these 108 nerve sutures.

Gosset[2] has reported 126 cases of nerve suture, of which 25 were performed by means of nerve grafting.

The results appear to him to be encouraging, and he ascribes the failure of suture "to the fact that nerve resections have been too restricted, and that the suture had involved sclerosed portions of the nerve, and for this reason was doomed to almost certain failure."

It is difficult therefore to formulate general and definite rules with regard to operation on nerves; one can only state what are the indications for operation, the most favourable period for surgical interference, and the clinical and histological findings in those cases which have resulted in failure. In short, though some definite points have emerged from the experiences of surgeons and neurologists during the last two years, the problem of surgical treatment of nerve injury is not solved in all its details. Nothing would be less scientific or more risky in the present state of our knowledge than to lay down definite axioms, always a dangerous thing in clinical medicine. The chief concern of the surgeon should be to respect the continuity of the nerve, not to form resections too hastily, and always to bear in mind the delicacy of nerve tissue, and the difficulty of its repair.

On the other hand, there is no justification for

[1] Tinel, *Les blessures des nerfs.* Paris, Masson, 1916.

[2] *Société de Chirurgie de Paris.* Meeting held April 12, 1916. *Bulletin et Mémoire de la Société de Chirurgie*, April 8, 1916, p. 971.

regarding as incurable nerve lesions more than a year old which have been operated on properly, even if they do not yet show definite signs of regeneration. Time may bring about unexpected cures. Therefore, when *a medical board has to make a decision,* a certain caution should be exercised in prognosis. The condition should be said to be permanent only in the minority of cases. The only fair and wise procedure is to propose an allowance which may be renewed after a long period (two years).

Indications for Operation. Technique

Just as each nerve reacts to injury in a special manner, so it seems to exhibit some peculiar process of regeneration. This individuality in the nerves from the point of view of their regeneration is an undoubted fact, but is as yet only imperfectly understood.

Exploratory Incision.—The first question is to know whether exploration is necessary, and how long after the wound the exploratory incision should be made. When the surgeon is sure of his methods there is no harm in exploration, and besides, it is not necessary to perform it in all cases.

Experience has taught us that a *very great number of nerve injuries* (*more than* 50 *per cent.*) *are cured spontaneously without any intervention.* It is therefore necessary first of all to find out by repeated and careful clinical examination whether the lesion is a slight one, which will probably undergo spontaneous regeneration.

Clinical examinations will have to be made at frequent intervals in order to arrive at a conclusion with the least loss of time.

In *complete paralysis of the musculo-spiral or of the external popliteal,* if the clinical signs remain severe four or five months after injury, and if the nerve shows no sign of regeneration, it may be assumed that there is an indication to interfere and to examine the appearance of the anatomical lesion.

If the nerve is found to be divided or completely crushed, suture performed directly after resection generally gives the best results.

With regard to injuries of the *ulnar*, the fifth month is also the maximum period of time to be allowed before exploratory incision should be made, when signs of a severe lesion are persistent.

This nerve is slow to regenerate after suture or resection, and it is important not to allow too much time to elapse before operating.

The *median* and *great sciatic* regenerate unsatisfactorily, slowly, and in a variable manner. Spontaneous regeneration never takes place in them in less than eight to ten months after the wound, but when these nerves are divided, crushed, or severely injured, their regeneration is particularly protracted. Thus, when clinical examination reveals persistence of the *signs of a severe lesion*,[1] the fourth month should not be allowed to elapse before interfering.

Naked-eye Appearance of the Injured Nerve.[2]—Inspection of the nerve is an important source of information from the point of view of surgical treatment. As the result of their histological researches, Pierre Marie and Foix have shown the way in which the appearance of a nerve should be interpreted. "We think," say these authors, "that between the anatomical condition as revealed by the simple naked-eye examination performed by the surgeon, assisted by the neurologist, and the histological structure there exists a sufficient resemblance to enable one to compare one with the other, and to draw from them practical conclusions." [3]

Complete Section, Suture, Grafting.—In the case of complete anatomical section, with separation of the two ends, the lower extremity of the upper neuroma will have to be pared away. This extremity is formed by fibrous tissue which opposes the advance of the

[1] See chapter on "Signs of Severe Lesions," p. 283
[2] See chapter on "Macroscopical Lesions," p. 231
[3] *Presse médicale*, January 31, 1916, p. 41.

axis-cylinders. The resection must be sufficient and must go beyond the zone of indurated tissue without, however, going as far as the perfectly normal part of the nerve. Excessive shortening of the central end would necessitate pulling on the nerve to allow suturing, or would require the use of a nerve graft, which is not to be recommended. Paring will be all the more necessary because the extremity of the central

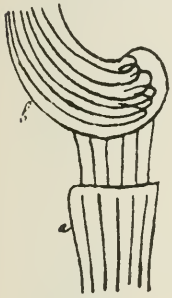


Fig. 25.

Torsion with horizontal direction of the fibres.

(After Pierre Marie and Foix, *Revue Neurologique*, Jan. 1916.)

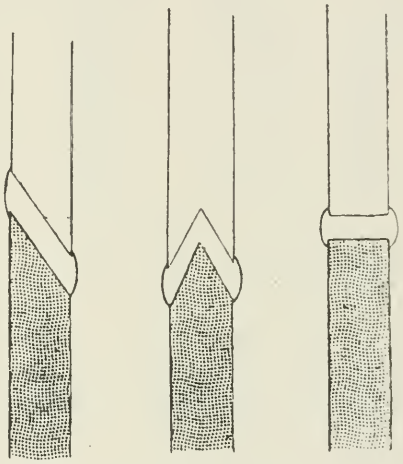


Fig. 26.—Different methods of end-to-end suture.

(After J. Sicard, *Paris Médical*, Feb. 19, 1916.)

segment often shows a kind of torsion with a horizontal arrangement of the fibres, which constitutes an impenetrable obstacle to the regenerating axis-cylinders.

The upper part of the lower pseudo-neuroma (peripheral end) must also be resected, since it is formed of fibrous connective tissue.

Once these two extremities have been freshened, they must be *sutured end to end* (fig. 26). The following is the method used by Gosset: Approximate the two extremities by catgut, then unite the neurilemmas by several fine silk sutures, taking care not to draw the two cut ends too tight, so as to

facilitate the advance in a straight line of the axis-cylinders of the central end (Nageotte).

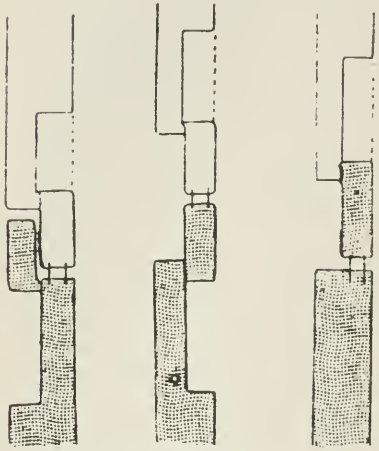


Fig. 27.—Several illogical and defective methods of nerve suture. (After J. Sicard.)

Any *operation must be rejected as illogical which involves any doubling over of one or other of the nerve segments, also bayonet suture, and especially the grafting of the central end on to a healthy neighbouring nerve, or the peripheral end on to a neighbouring mixed nerve* (fig. 27).

Grafting. — In all those cases in which the interval between two extremities of the nerves is too wide to allow of direct suture, the two ends can be joined together by the interposition or grafting of a segment of another nerve.

A small piece of a sensory nerve is taken and placed between the two ends of the cut nerve and sutured as before (fig. 28).

Fig. 28.—Nerve graft. (After J. Sicard.)

The results of nerve grafting which *a priori* appears to be a logical procedure, cannot yet be appreciated, since regeneration is beset with difficulties and takes place but slowly. What happens to this nerve segment deprived of all blood-supply and used as a scaffold? J. Sicard has suggested *neuro-vascular auto-grafting* by transplanting the segment of a neighbouring nerve while preserving a large pedicle of vascular and connective tissue.

This procedure would be applicable in all cases

where there was a healthy sensory nerve in the neighbourhood.

Lateral Notch. Preserve the Continuity.—The lateral notch should be treated according to Gosset's method. *Preserve the bridge of nervous tissue*, pare away slightly all the wall of the notch, and suture the two lips end to end (fig. 29).

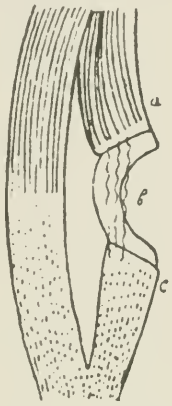


FIG. 29.—Lateral notch treated according to Gosset's method.

Crushing, Pseudo-continuity, Resection.—When the nerve is very comminuted, much torn, crushed, adherent to the neighbouring fibrous tissue, or imprisoned within a callus (as very frequently occurs in lesions of the musculo-spiral nerve), it often appears at the site of lesion as a flattened fibrous band situated between two swellings on the nerve and of a greater length than the nerve-trunk itself should be.

These cases are nearly always examples of complete section of the nerve with pseudo-continuity, the tissues between the two swellings consisting of fibrous and muscular tissue only. Under these conditions the only logical operation is *resection* of this bridge of tissue with freshening of the two extremities of the nerve and end-to-end suture.

But the naked-eye appearance of a crushed nerve is not always so characteristic, and does not allow us to conclude definitely in every case that the nerve is absolutely interrupted. This all goes to prove that repeated and careful clinical examinations are essential. It cannot be repeated too often that the neurologist and the surgeon must not forget *that very frequently injured nerves regenerate spontaneously* (about 70 per cent. according to certain statistics).

Resection must therefore only be performed when the cause of the condition is thoroughly understood—that is to say, when all the symptoms of a severe lesion are present, and when repeated clinical examinations

have not shown any sign of sensory electrical or motor recovery.

Pseudo-Neuroma of Attrition: Liberation: Hersage.— In the case of a pseudo-neuroma of attrition, the most frequent lesion of all, *the continuity of the nerve must not be interfered with;* it must only be freed, and when the surgeon is able to do so, he must attempt slight *hersage* of the neuroma in the line of the axis of the nerve by trying to incise the fibrous tissue between the nerve bundles without dividing them.

The process of freeing the nerve should always be carried out, whatever the nature of the operation performed on the nerve; it is also necessary so as to enable the operator to see the state of affairs and to judge of the condition of the nerve-trunk.

It is a good plan, then, to place the nerve among healthy muscular or aponeurotic tissue in order to separate it from the layer of bone below and from the skin above.

Various methods have been successively recommended for isolating the nerve from the neighbouring blood-soaked tissues, *e.g.* by healthy muscular or fatty tissue, a leaflet of peritoneum, the wall of an artery or vein, winding a thread of catgut round the injured nerve, etc.

But none of these methods seem to have given very satisfactory results. The essential is to follow several rules which are both logical and efficacious, viz. careful hæmostasis, washing the operation wound with normal saline in order to prevent desiccation of the nerve tissue during the operation, and to remove all the tissue débris, which would result in keeping up a subacute inflammation ending in the formation of a fresh fibrous cicatrix, and lastly, to place the nerve in a bed of healthy muscular or aponeurotic tissue.

The surgical treatment of *lesions of the brachial plexus* must be considered separately. We have drawn attention to the fact that lesions of the plexus tend to recover spontaneously. Nevertheless, when

at the end of a few months one or more of the nerve-trunks show signs of permanent physiological interruption, the time has come for surgical interference.

After making an extremely careful clinical examination and an exact localisation of the lesion, the surgeon should proceed to inspect the nerve-trunks and try to perform suture in those cases in which there is complete section.

Localisation on a diagram is absolutely necessary, not only for the incision of the superficial layers, but also to serve as a guide to the intricacies of the nerves constituting the plexus. Electrization will often be of great help during the course of the operation.

Operations without apparent Success. Causes of Failure.—We do not know if certain nervous lesions which have been judged incurable may not subsequently end in recovery. But when a suture has not given the hoped-for results even after several months, what are the causes to which one can attribute this delay in the regeneration of the nerve? Is the failure temporary or permanent?

When the operation has been performed too late, one can lay the blame on an ascending lesion of the nerve fibres of the central end. Degeneration of the peripheral end and of the muscles may also be incriminated.

In cases of sutures or resections performed early, and in which the post-operative history has followed the normal course, we must lay the blame on ascending degenerative lesions of the central fibres,[1] lesions

[1] The following description of these lesions is given by Pierre Marie and Foix: "Lesions of interstitial neuritis with islands of inflammation, lesions of parenchymatous neuritis with pronounced degenerative changes and descending and retrograde alterations of the axis-cylinders, medullary sheaths and sheaths of Schwann, excessive vascular changes with very marked endarteritis of the medium and small-sized vessels, perineural adhesions which are often hæmatomata secondarily organised by the sclerotic tissue, and neuro-muscular adhesions, which are frequently seen and important to recognise, since it emphasises the practical rule not to leave a nerve in contact with a bleeding muscular surface. All these changes are obviously in part responsible for the slow progress of functional repair," *Presse médicale*, No. 6, January 1916.

which reach a far higher level in some cases than is shown by the naked-eye appearance of the induration to be felt in the swelling on the upper segment. The interval between the two extremities must also be borne in mind.

Sutures performed by pulling on the nerve or the interposition of a nerve graft have far less chance of producing regeneration than end-to-end suture of two segments in close contact from the first.

The subsequent process of cicatrisation varies according to the case. Subacute inflammation of the section may arise, resulting in a fresh fibrous encystment of the central end, and possibly also of the peripheral end, thus creating a fresh obstacle to the advance of the axis-cylinders; finally the individuality of nerves from the point of view of their regeneration must be determined by the quality of their fibres and their intra-truncular arrangement.

The observations of Pierre Marie and Meige, therefore, possess an important practical bearing, and it will be very interesting to elaborate and complete them.

We have already seen that clinical examination of the median and sciatic nerves showed that they contained a very great number of sensory, vasomotor, secretory, and trophic fibres. Now if it is always preferable from the point of view of eventual motor recovery to bring into apposition the two ends of a cut nerve in such a manner that the motor fibres of the central end destined for a certain muscle should follow the sheaths of the lower end destined for this same muscle, all the more so is it necessary that motor fibres should not end in sensory areas, or *vice versa*.

The fibres constituting the posterior roots of nerves are more numerous than those coming from the anterior roots. On the other hand, certain nerves, such as the median and the sciatic, appear to contain a very great number of sensory, vasomotor, or secretory filaments. This explains how easy and unfortunate

it is for motor fibres to stray into sensory sheaths (Foix).

Foix has suggested the following hypothesis in order to explain the ease with which the musculo-spiral nerve regenerates, and the frequent failure of sutures of the median and ulnar nerves. The musculo-spiral gives off most of its sensory fibres very high up. The median and ulnar, on the other hand, have a large sensory area situated lower down, and their sensory fibres remain right up to the end united with the motor fibres.

The result is that paradoxical regeneration of the motor fibres in sensory sheaths is much more likely to take place in them, and if the many obstacles to satisfactory progress after suture be borne in mind, it is easy to understand why motor regeneration, which is relatively frequent in the musculo-spiral after suture, appears to be rare in the case of the ulnar and median.[1]

If to all these causes of failure be added other individual factors opposing regeneration, such as age, certain dyscrasias, and especially alcoholism, it is clear that regeneration after suture is not without difficulty. There is an obvious danger in deliberately resecting when the continuity of the nerve is preserved, and there is a possibility of interrupting a process of regeneration. It is impossible to tell if the resection and suture are not going to impair the regeneration of the nerve and end in complete failure.

Before closing this paragraph we would like to draw attention to two points: *What views should be held about immediate sutures? Must we refrain from operation through fear of suppuration in the wound which always takes place at first?*

We have been able to follow several cases of nerves which had been sutured immediately (two to eight days after the wound). Regeneration occurred invariably, and even more rapidly, it seemed to us, than

[1] Foix, *Société de Neurologie*, meeting held May 4, 1916. *Revue Neurologique*, June 1916, p. 904.

in other cases (especially in wounds of the sciatic). Other authors, such as Mendelssohn,[1] Braquehaye,[2] and Carrière,[3] have drawn attention to rapid regeneration after immediate suture.

It would be desirable for cases of this kind to be carefully studied. Surgeons at the front seem perfectly right in suturing immediately if the nerve is obviously cut. "Nerve suture, if done immediately after receipt of the wound," says Prof. Pitres,[4] "is a logical operation frequently of use, because by obviating the separation of the lips of the wound and the exuberant production of more or less tough connective tissue it facilitates the passage of the young fibrils of the central end towards the peripheral end of the nerve. But however easily and thoroughly this operation is performed, it does not prevent degeneration of the fibres of the peripheral segment.

"Late suture seldom gives favourable results, because it cannot be performed without causing a profound disturbance in the growth of the fibres which takes place at the extremity of the central end. *A fortiori*, is resection of this central end contra-indicated except in rare cases. It is seldom of use and generally harmful, because it destroys the whole histological process which has taken place in the zone of growth of the new axones."

The last point to settle is as follows: "*What views are to be held as to the immediate and rapid return of motor and sensory functions of a nerve after suture?*"

Personally we have never observed instances of this kind, and we do not know of any neurological communication describing such cases.

Pitres thinks that in view of the inevitable degeneration of the lower segment, even when sutured

[1] *Société de Neurologie*, meeting held December 2, 1915. *Revue Neurologique*, November–December 1915, p. 1285.

[2] Braquehaye, "Section du nerf médian droit. Suture immédiat du nerf. Retour partiel des fonctions," *Réunion médicale de la VIe armée*, November–December 1915.

[3] *Rapport mensuel du Service de Santé*, May 1916.

[4] *Journal de Médecine de Bordeaux*, December 1915.

immediately, "the opinion that occasionally immediate and ultra-rapid functional return of a divided nerve occurs is ill-founded, and rests on errors of interpretation. Among the observations which have been brought forward in its favour, there is not one which can bear searching criticism."[1]

Mme. Dejerine[2] thinks that "rapid recovery of motility occurring one or two days after suture of the nerve is absolutely incompatible with clinical, anatomical, and pathological experience. It has neither been observed after primary sutures performed immediately after injury, nor in secondary sutures performed at a later date. . . .

"After nerve section the peripheral end of the nerve always degenerates throughout its whole extent.

"If 'rapid' motor recovery does not exist from a histological point of view, how are we to interpret the so-called rapid recovery which has been clinically confirmed a day or two after nerve suture? When these cases are critically investigated they are always found to be inaccurate and to lack clinical precision."

Surgical Treatment of Painful Forms of Nerve Lesions

We have seen in studying the symptomatology of painful cases that there is often a development of ankylosis and retraction rendering the use of the limb absolutely impossible. Apart from the gloomy outlook of such a condition, the intensity of pain which is unrelieved by internal medication or external application suggests surgical intervention. Simple *freeing* of the nerve, which seems the only method to follow in these cases where the anatomical lesion is confined to a thickening or induration of the nerve, does not give satisfactory results.

[1] *Loc. cit.*

[2] *Société de Chirurgie*, December 1915.

Resection of the nerve followed by immediate suture above the wound has not always resulted in complete and permanent abolition of causalgia. On the other hand, section of a mixed nerve, which is apparently only slightly injured, is always a serious measure, since motor recovery may suffer through it.

Sicard conceived the idea of using the method of *alcoholisation of the nerve-trunk*, which he had already employed in trigeminal neuralgia. The results obtained by him seem to be promising.

His method is as follows : under general anæsthesia the injured nerve-trunk is exposed, freed from the surrounding cicatricial tissue, and then 1 cubic centimetre of sterilised alcohol at 60° is injected with a fine needle. The injection must be made 2–3 centimetres above the lesion. When successful, a small œdematous swelling appears, and the nerve-trunk at once assumes a characteristic white appearance. The alcohol should be preserved in ampoules sterilised by tyndallisation in order to maintain its strength.

Pain vanishes instantaneously, and, on the other hand, the alcohol is said to interrupt motor conductivity for only a relatively short time (six to ten months).

Alcoholisation of the nerve-trunk, which alleviates the pain, is also said to stop the progress of ankylosis, and prevents retractions and dystrophy of all the tissues of the affected limb.

We have elsewhere recorded a case of causalgia of the median nerve, operated on by Gosset, who performed hersage on the swelling of the nerve-trunk. This hersage, which doubtless acted by tearing apart the nerve filaments without dividing them, brought great and progressive relief from pain, so that in eight to ten days the patient hardly suffered at all.

Another form of treatment of these nerve injuries has been advocated by Leriche.[1] It consists in exposing the brachial artery (for causalgia of the median) and excising all its sympathetic and connective-tissue

[1] *Presse médicale*, April 20, 1916.

sheath for about 12 centimetres. It is said that the resulting freedom from pain is considerable.

Other investigators have tried the same method on the popliteal artery for causalgia of the sciatic nerve. It is, however, better to wait before expressing an opinion about the efficacy of these operations.

CHAPTER VII

PHYSIOTHERAPEUTIC TREATMENT OF INJURIES TO NERVES

Physiotherapeutic methods hold the first place in the treatment of nerve lesions. Occasionally they are used as a complement to surgical treatment, and sometimes alone, in those numerous cases in which the nerve lesion tends to heal without operation.

Given the long period required by the evolution of a nerve lesion even in cases of physiological interruption of moderate degree, it is essential to prevent degeneration of the paralysed muscles, to keep the circulation of the affected limb active, and to prevent rigidity of the joints and vicious attitudes.

Massage, *movement*, *electrotherapy*, and the wearing of suitable *appliances* help in attaining this end.

Massage and Mobilisation

Hand massage and vibratory massage should be employed daily, and often for many months. The essential complement to this treatment is *passive movement of the joints*. Rapid muscular atrophy is thereby avoided, absorption from the infiltrated tissues is favoured, and rigidity of the articulations prevented. Passive movement can only be of short duration after the daily massage, and is not sufficient in cases of multiple ankylosis.

Frequent treatment by *active mechanotherapy* and *gymnastics* should be employed as well. The patient

should also be strongly advised to move the different segments of the affected hand several times daily with his sound hand.

This unceasing collaboration of the patient and the doctor is the best of all methods of physiotherapy.

In painful forms, with a tendency to articular rigidity and deformity, we must have recourse to massage and movement as soon as the pain subsides a little. Since water is the favourite element with patients of this kind, they should get accustomed to use their flexor muscles by making them squeeze a wet sponge, and by *having them massaged lightly under water* (Meige).

We will not describe the various installations and appliances used in mechanotherapy, which are sometimes elaborate, sometimes simple, and mostly useful, provided that they are thoroughly adapted for their purpose. It is very important that the physician should arrange about their application himself, and that he should supervise this part of the treatment.

When nerve regeneration has in a great measure taken place, or when, on the other hand, regeneration does not seem likely to occur, it is useless to pursue mechanical or electrical treatment any further, since it will be superfluous. It is very important, however, to massage and use the battery on an injured limb during the first few months after the wound, as regeneration of the nerves and muscle is assisted thereby. When clinical examination has shown the persistence of the signs of a severe lesion, such as complete paralysis, and when an operation has been performed, massage should be resumed, as it will then help in regeneration.

When signs of recovery begin to appear, it is often better to send a patient back to civil life for several months, by temporarily discharging him from the service. The patient who has to earn his living always hastens his recovery.

But it is absolutely necessary to discharge any soldiers who have suffered from nerve lesions from

a year to eighteen months, and who have been operated on more than six months previously, and who have had an uninterrupted and appropriate physiotherapeutic treatment for more than one year.

If recovery is to appear at a later date, the exercise of a trade, we repeat, will be better for him than rest.

If the paralysis is permanent, and if regeneration appears out of the question, it would be wrong both to the individual and to society to prolong the illusory treatment.

Electrotherapy

The currents used in electrical treatment of paralysis of the nerves are of two sorts: the constant or galvanic current, and the induction or faradic current.

1. The *faradic current* is produced by an apparatus similar in structure to the Ruhmkorff coil. The majority are composed of a primary coil enclosing a core of soft iron. The extremities of this coil are joined to the poles of a battery. (A portable apparatus contains two or three dry cells.) Over this coil another coil, called the secondary coil, is made to slide. By means of an interruptor variations are produced in the continuous current which flows through the primary coil. Each wave of closure or opening thus produced in the primary circuit develops an induced electromotive force in the secondary circuit. It is this induced current which is used in electrotherapy. The faradic or induced current is still graduated and measured in quite an empirical way by more or less sliding the movable coil over the primary coil.

2. The *galvanic* or *continuous current* is furnished by batteries composed of zinc chloride or, better still, of sulphate of mercury. The batteries in general use, consisting of thirty-two to forty cells, are sufficiently powerful.

The galvanic or continuous current is graduated by means of collectors, and is measured by means of a small apparatus called *milliampèremeter*.

In order to introduce the current into the organism, appliances called electrodes are used. The best electrodes are those of cotton-wool. They are made of tin plate of variable size, covered on one side with several layers of cotton. The whole is enclosed in a covering of tightly stretched gauze.

Before being applied the electrodes should be thoroughly soaked in slightly salt, tepid water.

Each sitting should last ten to fifteen minutes, and should be repeated every day or every second day.

"**Painless Paralysis.**[1]—When electrical examination has shown impairment of electrical contractility, appropriate treatment must be chosen for each particular case.

" Paralysis accompanied by partial and slight R. D., and which is not painful, should be subjected to the action of the galvanic current, together with a faradic current with slow interruptions (galvano-faradisation). Return of motility will thus be facilitated, and muscular atrophy prevented.

" Paralysis accompanied by well-marked partial R. D. or complete R. D. should be treated by the galvanic current.

" The rhythmical galvanic current should be used in painless paralyses, and those limited to a single nerve by localising the current on muscles supplied by this nerve ; movements which the patient cannot perform voluntarily will thus be easily elicited.

" But if too great diffusion of the current provokes movements in antagonistic muscles to the detriment of the injured muscles, rhythmical galvanisation should be replaced by the continuous current. Whatever may be the character of the continuous current, only the current from cells or accumulators should be

[1] We are much indebted to Dr Grunspan for this note on the electrical treatment of painful and painless paralyses.

used. The current supplied by the sector is a wave current, and therefore painful. Care must be taken to stuff the electrodes with several layers of cotton-wool soaked in hot water.

"**Painful Paralysis.**—The continuous current well applied is an excellent therapeutic agent; it stimulates vasomotor activity in the region to which the electrode is applied, awakens the excitability of the nerves, and exercises at the same time a sedative influence; it is therefore used with great success in painful paralysis.

" Under these conditions the negative electrode is placed on the point of emergence of the nerve, and the positive electrode on the hand if the patient is affected by painful paralysis of the upper limb, or on the foot if the paralysis is in the lower limb.

" The current must be made to reach the point of maximum intensity desired by proceeding to increase it gradually; it should seldom be below 50 milliampères, and the application should last at least fifteen minutes. The electrodes must be well applied, and fixed in order to distribute the current evenly and to prevent burns. Some painful paralyses resist the sedative action of the continuous current. In these stubborn cases excellent results are sometimes obtained by radiotherapy."

Electrical treatment, just like mechanical treatment (massage, gymnastics), must not be persisted in indefinitely.

If at the end of ten or twelve months there is no sign of recovery, it is best to send the patient back to civil life. If the injured nerve is going to regenerate, the muscular work which he will be obliged to perform in carrying on his trade and in his ordinary life will take the place of the benefit which he would derive from electrization for a few minutes at a time.

If the nerve is definitely lost, no physiotherapeutic treatment will prevent the complete degeneration of the paralysed muscles.

Radiotherapy

Radiotherapy has been mainly used in painful lesions of the nerves, and especially in wounds of the median and sciatic.

Cestan and Paul Descomps[1] first systematically used this method and obtained satisfactory results.

The technique is as follows: "1 milliampère with a hard tube (the hardness of the tube being essential). Rays VIII–IX. Aluminium filter of 1 millimetre. Distance of the anti-cathode from the skin 20 centimetres. One sitting per week lasting a quarter of an hour."

Mlle. Grunspan uses the following method, which had previously been recommended by Babinski for stubborn neuralgias.[2]

The point of emergence of the nerves is irradiated once a week for three weeks. In particularly painful neuritis of the median two irradiations are performed a week, one on the cervical roots, the other on the brachial plexus. This treatment is continued for three weeks, followed by a period of three weeks' rest, after which a fresh series of six applications can be repeated.

Pain can also be satisfactorily alleviated by heat in every form, such as hot-air baths, light baths, and diathermy.

Diathermy has a great advantage over the other two methods, in that it penetrates throughout the entire thickness of the tissues.[3] But this process demands expensive appliances, and necessitates skilled supervision throughout the whole application.

[1] Cestan and Paul Descomps, "La radiothérapie dans le traitement de certaines lésions traumatiques du système nerveux," *Presse médicale*, November 25, 1915.

[2] Babinski, "Spondylose et douleurs névralgiques atténuées à la suite de pratiques radiothérapiques," *Société de Neurologie*, May 5, 1908.
Babinski, Charpentier, and Delherm, "Radiothérapie de la sciatique," *Société de Neurologie*, April 6, 1911.

[3] M. Grunspan, "Essais de mensuration de la température réelle des tissus au cours des traitements par l'air chaud, la diathermie et l'électrocoagulation," *Revue de Chirurgie*, 1912.

CHAPTER VIII

ORTHOPÆDIC APPLIANCES

As soon as patients suffering from nerve injuries were admitted into the clinic of Prof. Pierre Marie, Henry Meige set about improving the functional disabilities and deformities produced by paralyses and contractures resulting from nerve lesions. With this end in view he proceeded to create a whole series of corrective appliances adapted to the peculiarities of each individual case, and frequently altered and improved them, his principal object being to simplify them and adapt them for daily use. In July 1915 he laid before the Neurological Society of Paris the essential conditions which these prosthetic appliances must fulfil.

"It is of the utmost importance," he said, "when a group of muscles is paralysed to oppose as soon as possible the ascendancy of the antagonistic muscles.

"Massage and movement have this end in view. This object should also be the initial aim of appliances; they prevent and correct vicious positions, and at the same time favour motor recovery.

"First of all, they lessen the resistance which the weight of an extremity, too heavy for the regenerating muscles, opposes to their first tentative contractions.

"Over and above that, the antagonistic muscles as soon as they are immobilised lose a part of their contractile force; they become accustomed to relaxation, and the struggle becomes less unequal with the muscular group, which is beginning to have contractions of its own."[1]

[1] Henry Meige, "Il faut favoriser les restaurations motrices à la suite des blessures des nerfs au moyen d'appareils appropriés," *Société de*

These appliances "should be easy to make, cheap, and light; they must not compress the tissues, and they should be as inconspicuous as possible."

"Each patient should have an appliance specially constructed for him, on account of the great individual variations in skeletal and muscular structure, the localisation and intensity of the paralyses, and the presence of complications in the bones, tendons, or blood-vessels, pain, etc.

"It is necessary that these appliances should be made to measure, tried on many times, modified according to individual peculiarities, and re-made as often as necessary."

"At the same time it is very important to regulate the time during which such an appliance is to be worn daily, since a certain period must elapse before the patient becomes accustomed to its use. At first it should be worn for a few hours only, the time being gradually increased until it is worn throughout the whole day.

"If the appliance causes any discomfort or pain, even if slight, it must be frequently altered until it is thoroughly comfortable and satisfactory.

"Inversely, as motor recovery progresses, the time during which the apparatus has to be worn is decreased, or another may be substituted if it is better adapted for the new state of the patient.

"In all cases frequent inspection is necessary to control the progress and check any faulty movements" (H. Meige).[1]

These appliances are specially useful in musculospiral paralysis and in lesions of the external popliteal to correct hand- or foot-drop.

All neurologists have exerted their ingenuity to remedy faulty positions of the limbs by more or less

Neurologie, meeting of July 29, 1915. *Revue Neurologique*, August–September 1915, p. 761.

Pierre Marie and Henry Meige, "Appareils pour 'blessés nerveux,'" *Académie de Médecine*, meeting of August 10, 1915.

[1] *Loc. cit.*

simple means, and it may be said that to-day in every neurological centre similar appliances are used, and that all are capable of useful service.

We cannot enumerate, far less describe, all of them. We only mention those which we saw used, and which were appreciated by the patient, since the satisfaction which an appliance gives to the patient is the best criterion of its excellence.

The Neurological Society of Paris has appointed a commission consisting of Souques, Mme. Dejerine, Henry Meige, Jean Camus, and Froment "to formulate the essential conditions which must be fulfilled by a prosthetic appliance for nerve injuries, and to state the indications for their use." The first two reports of this commission contain the following suggestions :—

"First of all, we think we ought to issue a warning against a certain number of errors committed in the conception and application of prosthetic apparatus for nerve injuries.

"These errors may have the most regrettable consequences.

"Occasionally the appliance does exactly the opposite to what we desire to effect.

"Thus immobilisation appliances are used in cases where movement is indicated, while in other cases appliances are used which are heavy and tend to cause excessive compression, thus favouring muscular atrophy and preventing motor recovery. Or an appliance which has been successfully used to treat a definite form of paralysis is wrongly applied to another type of paralysis, in which its action is useless, or even harmful. Another error is that patients suffering from hysterical paralysis are not uncommonly seen wearing appliances intended for organic lesions. Here the prothesis not only confirms an error of diagnosis, but, what is graver still, it tends to render chronic or even permanent disorders which by their very nature are essentially and rapidly curable."

Principal recommendations :—

1. Carefully select the points of support in order to avoid compression of muscles, vessels, or nerves.
2. Avoid all excess in correcting deformities.
3. Favour the play of the affected muscles and their antagonists, without allowing predominance of the latter.
4. Graduate the mechanical effects of the apparatus methodically.

" Two types of apparatus are to be considered :—

" 1. *Appliances for temporary incapacity :* these should be light, practical, easy to alter as progress becomes apparent, and specially designed to favour this progress.

" 2. *Appliances for permanent incapacity :* these are mainly intended to be of practical use, and should enable the individual to carry on his occupation. They must be strong, easy to put on and take off, and simple to clean."

Appliances intended to Correct Musculo-Spiral Paralyses

In musculo-spiral paralysis the most important aim of prosthetic appliances is to correct " an attitude of the hand which is not only ungraceful but impedes flexion of the fingers, which is generally well preserved. They also cause the swelling on the dorsal aspect of the carpus and the œdema of the metacarpus, which is so frequently seen, to disappear. In short, their effect is to oppose the tendency of the flexors to embody the whole motor activity and to retract " (H. Meige).

The simplest and the most practical appliances consist of a *palmar support* which prevents wrist-drop.

A simple aluminium splint bandaged to the forearm has been recommended by Tuffier.

We have found that the appliances made in the service of Prof. Pierre Marie at La Salpêtrière according to the directions of Henry Meige have given most satisfactory results.

The first model consists of an aluminium splint, which encloses the palmar aspect of the forearm, and is prolonged on to the palm of the hand by a sort of spoon-shaped continuation which is accurately adapted to its shape. This prolongation curves slightly back on the internal border of the metacarpus, and has a hook situated between the thumb and the index finger.

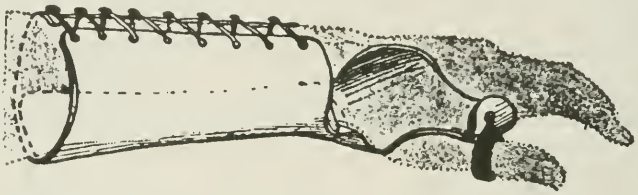


FIG. 30.—Applicance of Henry Meige (musculo-spiral paralysis). Leather cuff holding an aluminium trough, in which the hand rests horizontally, permitting flexion of the fingers. The thumb is supported by a ring of leather.

The appliance is surrounded by a sheath of leather, and is fixed to the forearm by a long cuff kept in position by a lace passing through eyes and hooks. The patient can easily put on or take off his appliance, which is also very inconspicuous (fig. 30).

The same apparatus can be modified in order to

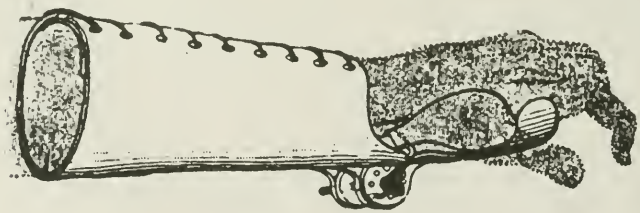


FIG. 31.—Articulated supporting appliance, allowing angle of inclination of hand on forearm to be varied (musculo-spiral paralysis, contracture of the flexors). (Henry Meige.)

correct stiffness of the wrist by adding a sort of joint between the forearm splint and the palmar spoon, whereby the inclination of the hand can be graduated and the hand can be maintained in the desired position (fig. 31).

Another type of appliance consists of an aluminium splint applied by means of a leathern cuff to the dorsal surface of the forearm. From the lower part of this splint four flat springs project, ending in rings, through which pass the first phalanges of the last four fingers.

The rigidity of the springs is sufficient to correct the wrist-drop, besides which it does not impede the action of the flexors of the hand and fingers, so that

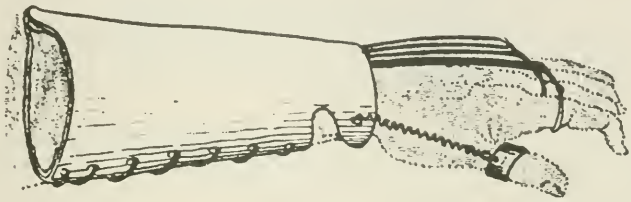


Fig. 32.—Spring appliance of Henry Meige, keeping the fingers and the hand horizontal (paralysis of the extensors of the wrist and fingers). The thumb is abducted by a ring of leather fixed to a spring catch.

the patient is able to perform all the movements of prehension (fig. 32).

Henry Meige has designed a still more simple and practical *apparatus with a palmar button* (figs. 33 and 34).

It consists of a metal splint, which is fastened to the lower third of the forearm by a leathern cuff. At the carpal border of the splint is fixed a slightly bent iron wire ending in a padded button, which rests in the hollow of the hand.

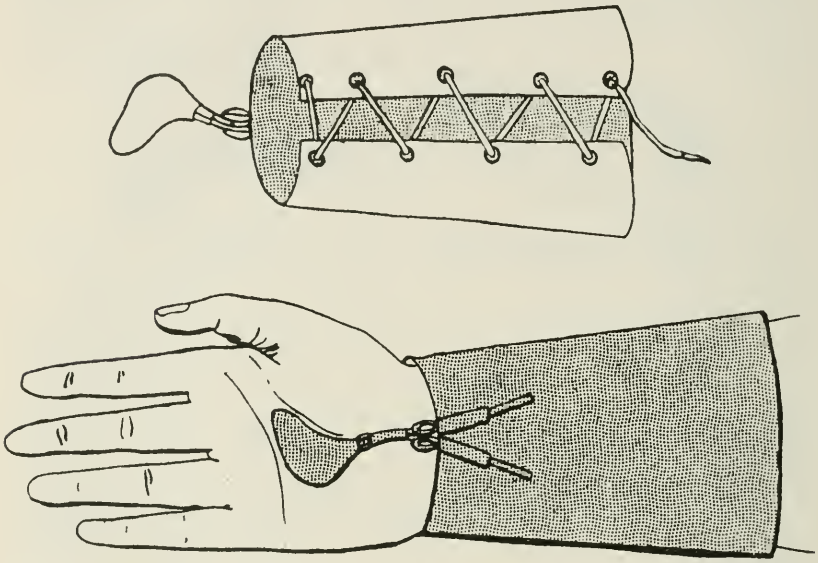
The iron wire is rigid enough to support the weight of the hand and to correct its tendency to drop ; it even allows the patient to lift heavy weights without the hand giving way, which is so troublesome in ordinary movements. It also has the great advantage of allowing complete flexion of the fingers and adduction of the thumb.

It can be made as resistant as desired, and is then most valuable for workmen.

It is also the most convenient appliance to write

with. Lastly, it is quite inconspicuous, and easily and cheaply manufactured.

The palmar button can be separated from the arm-piece, which enables the two pieces to be made in advance and to be fitted separately, according to the size and dimensions of the patient's hand and forearm.



FIGS. 33 and 34.—Appliance for musculo-spiral paralysis with palmar button. (Meige.)

To correct paralysis of the extensors and the abductor longus pollicis these appliances can be furnished with a leather ring fixed to a spiral spring intended to separate the thumb.

Another model has been used by Souques, Mégevand, and Donnet.[1] It consists of a leather arm-piece lined with chamois leather, surrounding the lower end of the forearm and lacing up on its anterior aspect.

At each side of the arm-piece an iron rod is fixed, articulating with another bent iron rod which unites at the palm with that of the opposite side. The articula-

[1] *Société de Neurologie*, June 29, 1916. *Revue Neurologique*, July 1916, p. 119.

tion between the two rods takes place at the wrist, and is homologous to it in its action. Successive

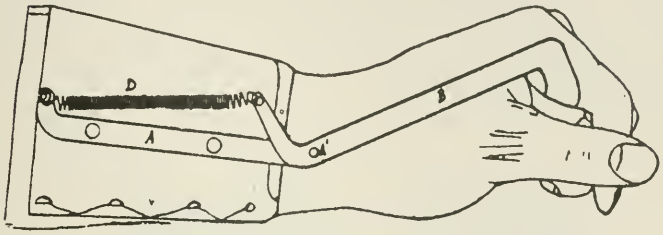


FIG. 35.—Appliance for correction of musculo-spiral paralysis (appliance at rest). (A. Souques, Mégevand, and Donnet.)

movements of voluntary flexion and mechanical extension can thus take place freely (figs. 35 and 36).

The appliance of Froment and Muller (figs. 37 and

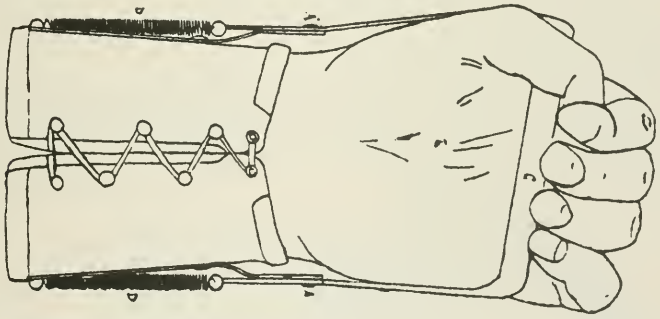


FIG. 36.—Appliance for correction of musculo-spiral paralysis (front view).

38) is of an analogous design, but can be locked, the hand remaining slightly hyperextended to allow heavy objects to be grasped; it is a good apparatus for working with.[1]

Other equally practical appliances are those of Privat and Belot (figs. 39 and 40), Léri and Dagnan-Bouveret (fig. 41), Mouchet and Anceau, Rieffel and Ripert.

The commission appointed by the Neurological

[1] *Revue Neurologique*, July 1916, p. 123.

Society to investigate prosthetic appliances for patients suffering from wounds of the nerves has directed its attention mainly to the requirements of working appliances in cases of musculo-spiral paralysis. The following are the principal remarks contained in their second report:—

"The dominant question is for the hand to find *a*

FIG. 37.—Appliance for permanent musculo-spiral paralysis seen from the upper surface. (Froment and Muller.)

fixed point in the apparatus which will oppose its tendency to drop, even during effort and when the object grasped is heavy.

"Can *an extensor spring supply this desideratum*? To this we have to reply in the negative. . . . Thus

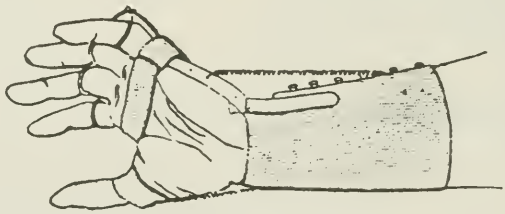


FIG. 38.—The same seen from the lower surface.

it has been found that some workmen take off their appliance to work.

"In distinct opposition to appliances designed with the object of re-educating the parts, we have here to supplement the spring by a catch *which arrests and maintains the hand in the most useful position.* This position is in general that of slight extension analogous to that in which the hand is placed when making an energetic grasping effort. *This catch is essential.*

"The following types of catch have been suggested:

"An elastic catch with a palmar button concealed in the hollow of the hand (Meige's model).

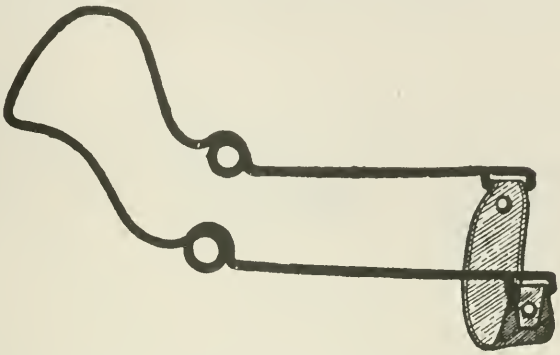


Fig. 39.—Prosthetic appliance for paralysis of the musculo-spiral and external popliteal nerves. (J. Privat and J. Belot.)

"A dorsal catch or extensor spring supplied with a catch (Camus's model).

"A catch hidden in the articulation on the sides of the wrist and leaving the palm of the hand quite free.

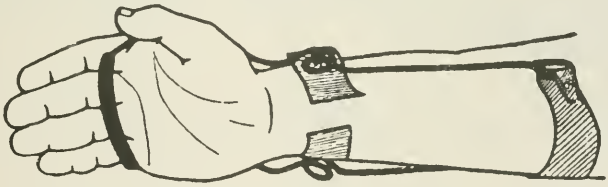


Fig. 40.—Appliance of Privat and Belot in position.

This catch may be fixed or adaptable to various positions (Froment and Muller, types A and B)."

"Other types of catch may be invented.

"The essential point is the presence of an efficient catch, and one which is concealed and leaves the hand free.

"In appliances intended for the use of working men and labourers, the aim is not so much lightness or concealment as strength, ease in putting on and

taking off and in cleaning it. The method of attachment to the forearm should also receive careful attention. Although a short arm-piece is for many reasons desirable, it is better to distribute pressure over a wider area than to have a localised and powerful

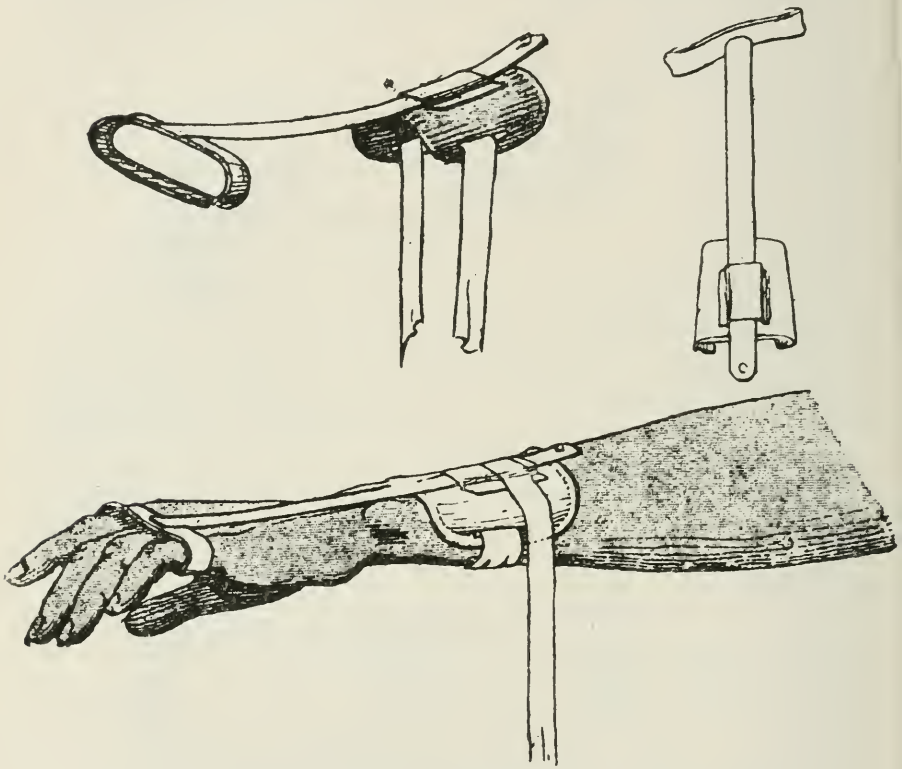


FIG. 41.—Appliance of Léri and Dagnan-Bouveret for musculo-spiral paralysis.

constriction, for it is to this part that is transmitted the resistance to the movement of flexion which is arrested by the catch.

"For the *business man*, on the contrary, a light, inconspicuous, and convenient appliance is essential, one with which he can write and draw. Here the problem is simpler, and is partially solved by appliances already in use. It should be noted that the

metal pieces placed on the ulnar border of the hand are very inconvenient when writing."

All these appliances aim at correcting paralysis of the extensors of the wrist (radial muscles), but they do not compensate for paralysis *of the extensors of the fingers.*

Naturally attempts have been made to correct this latter disability, and by the aid of flat or spiral springs, or elastic tractors with one end fixed to the wrist and the other end to the phalanges, this has been fairly satisfactorily accomplished.

An appliance of this sort had originally been invented by Duchenne (of Boulogne). Mme. Dejerine has had one made in which the digital attachments are very well thought out. Sollier, Cestan, Chiray, etc., have devised other models which certainly give good results. The latest type (that of David and Cateau), made at the Grand Palais, is remarkably adaptable.

Generally these appliances with digital adaptations fulfil their aim fairly well, which is to compensate for the paralysis of the extensors of the fingers, but they are more complicated and costly, and they are also more cumbrous. They are specially useful to patients who have to perform delicate movements with their fingers.

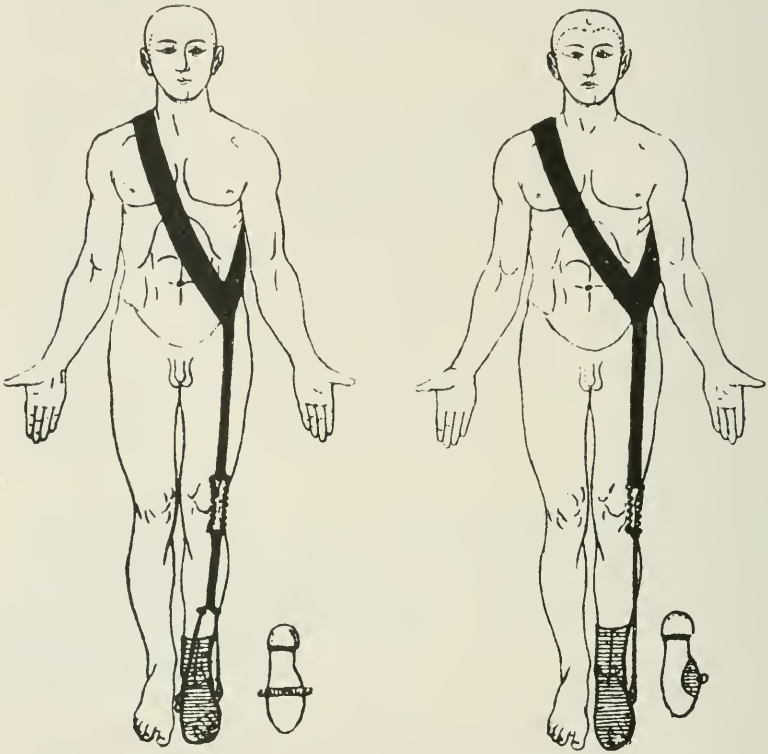
In the majority of cases the simplest appliances should be preferred, such as Henry Meige's palmar button apparatus.

Appliances for Sciatic Paralyses

Shoulder-belt Appliance of Henry Meige.—The principle of this apparatus is as follows:—The affected foot is raised by traction starting from the shoulder of the opposite side by means of a belt slung over the shoulder and prolonged downwards by an elastic suspender and fixed below to the sole of the boot of the paralysed foot (figs. 42 and 43).

If there is paralysis of all the leg muscles, the lower

attachment is effected by two cords fixed to each side of the sole or by a stirrup. This apparatus has the advantage of ensuring the raising of the foot, since its



A B

FIGS. 42 and 43.—Appliances for foot-drop. (Henry Meige.)

Sling passing from the opposite shoulder to the affected foot and holding up an elastic suspender fixed to the sole of the boot. A, by two attachments (paralysis of all the muscles raising the foot); B, by one attachment to the external border (paralysis of the external popliteal).

drop is very inconvenient when walking, and of preventing a steppage gait.

In paralysis of the antero-external group of muscles a single attachment to the outer border of the boot is sufficient. As a rule, to enable the foot to be firmly

planted on the ground, it is advisable to raise the

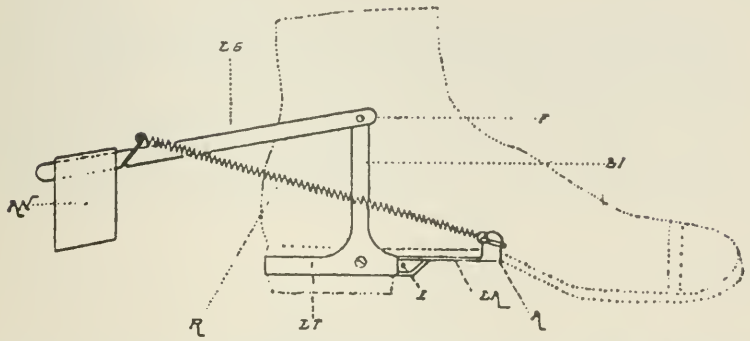


FIG. 44.—Prosthetic appliance for paralysis of the sciatic nerve (appliance ready to be put on to foot). (Souques, Mégevand, and V. Donnet.)

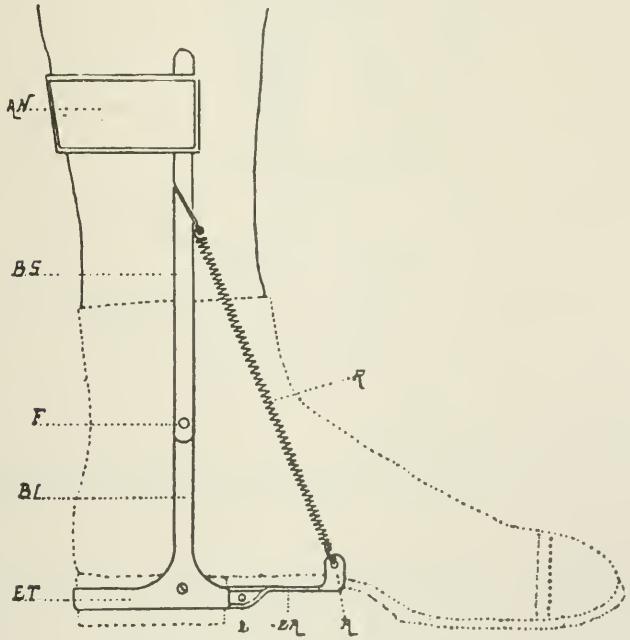


FIG. 45.—The same appliance fitted on.

external border of the boot by means of a layer of leather about 1 centimetre thick.

This raising of the sole is sometimes enough to

correct the defect in gait when the foot-drop is not too marked. Its use is still required for some time even after discarding the other corrective appliances.

Appliance of Souques, Mégevand, and Donnet.[1]—The two lateral springs which constitute the essential part of the apparatus communicate to the foot a movement of flexion when it leaves the ground. By flexing the foot on the leg, this appliance maintains it in

FIG. 46.—Appliance of Privat and Belot for paralysis of the external popliteal.

a good position without causing any false movements of rotation or distortion.

The half ring of the upper portion is simply applied to the calf and only exerts slight pressure (figs. 44 and 45).

Appliance of J. Privat and J. Belot.[2]—The apparatus is of simple construction, and consists of a steel spring fixed underneath the sole, and of a leather strap which is tightened behind the calf in order to produce tension of the spring (figs. 46 and 47).

[1] *Revue Neurologique*, November–December 1915, p. 1253.
[2] *Ibid.*, July 1916, p. 120.

Similar useful appliances have been thought out and made by A. Léri, Sollier, etc.

Of all these appliances preference must be given to that of Souques, Mégevand, and Donnet, which corrects foot-drop remarkably well, and which is in addition strong, cheap, and inconspicuous ; all our patients fitted with it have been well satisfied.

The shoulder-belt appliance of Henry Meige is

Fig. 47.—Same appliance fitted on.

especially indicated in cases where there is extreme tenderness or a painful wound of the calf.

Appliances for Paralysis of the Median

Appliances of this kind are far less numerous. This can be explained by the fact that paralysis of the median involves a far less noticeable deformity than does paralysis of the musculo-spiral, the lesion only becoming noticeable when the patient tries to perform movements of grasping.

In order to remedy this inability to oppose the thumb to the fingers, J. Froment and Wehrlin[1] have

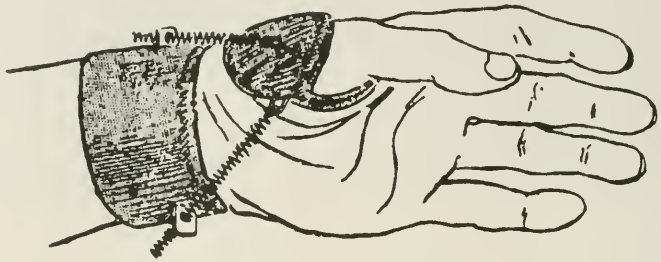


Fig. 48.—Appliance of Froment and Wehrlin. Semi-diagrammatic drawing. The position of the dorsal spring on the forearm has been slightly lowered for it to be visible in this position of the hand and give a view of the mechanism as a whole.

devised the following appliance, which fulfils its purpose well and is simple in construction.

It consists of a leather cuff round the wrist and of a ring worn round the thumb. The ring is connected with the cuff by two springs, one of which is inserted on the outer border of the metacarpo-phalangeal joint of the thumb, and is directed towards the pisiform bone, being attached to the cuff at the inner border of the wrist. This spring is intended to replace the paralysed opposing muscle, and should be well on the stretch.

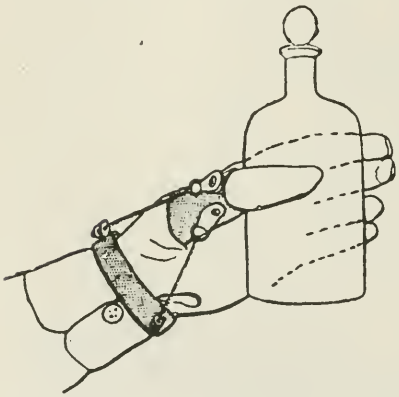


Fig. 49.—A new type of prosthetic appliance for paralysis of the median. (J. Froment.)

A second spring is attached to the ring on the dorsal aspect of the metacarpo-phalangeal joint and to the cuff on the dorsal aspect of the wrist in the neighbourhood of the posterior border of the radius. This spring

[1] *Revue Neurologique*, November–December 1915, p. 1233.

plays the part of antagonist, and should therefore not be much on the stretch (fig. 48).

Froment[1] has modified this appliance in such a manner that the springs, instead of being attached to the base of the first phalanx of the thumb, are inserted into its upper extremity (fig. 49).

This arrangement allows extension of the last phalanx of the thumb, and thereby effects a more perfect and efficacious opposition to the fingers.

Appliances for Ulnar Paralysis

An appliance for ulnar paralysis has been invented by Cuneo and Rolland with the object of "preventing or correcting vicious attitudes and secondary deformities, which are often the cause of the poor results of operations for compression or division of nerves."

"I am able to state," says Cuneo, "that the use of corrective appliances for these deformities not only obviate additional operations, but also considerably hasten the return of voluntary motility."[2]

Two kinds of cases may be met with :—

(*a*) *Ulnar Paralysis with Claw-hand.*—The appliance will consist of a forearm-splint keeping the hand in hyperextension. To this splint two arched supports are fixed. The first one is ventral and holds the series of elastic tractors intended to produce flexion of the fifth, fourth, and, if desired, third finger as well (first action of the interossei). A second series of tractors fixed to a second arched support will produce extension of the second and third phalanges (second action of the interossei).

(*b*) *Complete Claw-hand* due to section of the ulnar and median at the wrist. A similar appliance, but furnished with eight instead of six tractors, and having in addition a special tractor for the thumb, will supply all the movements needed.

In spite of all these interesting attempts, prosthetic

[1] *Revue Neurologique*, July 1916, p. 125.
[2] *Bulletin de la Société de Chirurgie de Paris*, March 15, 1916 (No. 10).

appliances for ulnar paralysis are at present very unsatisfactory, because in the majority of cases there are obstinate retractions of the tendons which frequently become irreducible.

Appliances for Paralysis of the Upper Brachial Plexus

The lesions of the upper brachial plexus leave the motility of the hand practically unaffected, but the

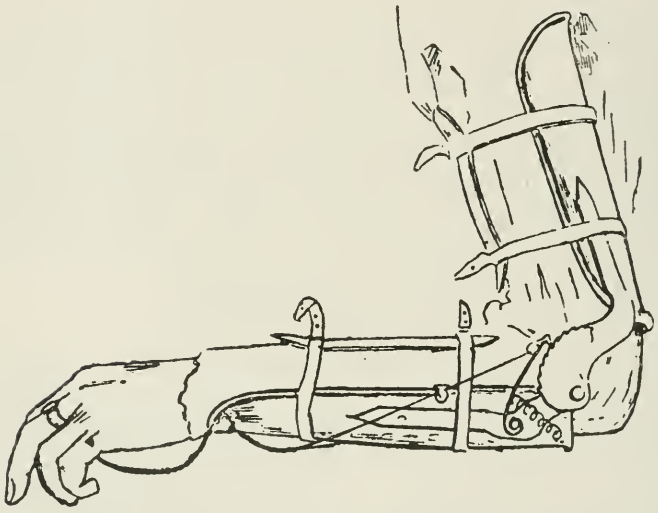


Fig. 50.—Position of flexion allowing the patient to use his hand. The angle of flexion can be varied at will, the forearm preserving the position in which it has been placed. (J. Dagnan-Bouveret.)

hand is useless owing to permanent extension of the forearm on the arm.

It is in order to get a useful flexion of the elbow, which, on the other hand, must not be permanent, and can be varied at will, that Dagnan-Bouveret has invented an ingenious apparatus, which he describes as follows :—

" It consists of an arm-splint and a forearm-splint united at the elbow by a double lateral hinge joint.

On the pivot of each of these hinge joints, and fixed to its upper branch, there is a segment of a serrated wheel. The lower branch has a catch which bites on this wheel, and is kept in contact with it by a spiral spring.

" On the other side of the spring the catch has

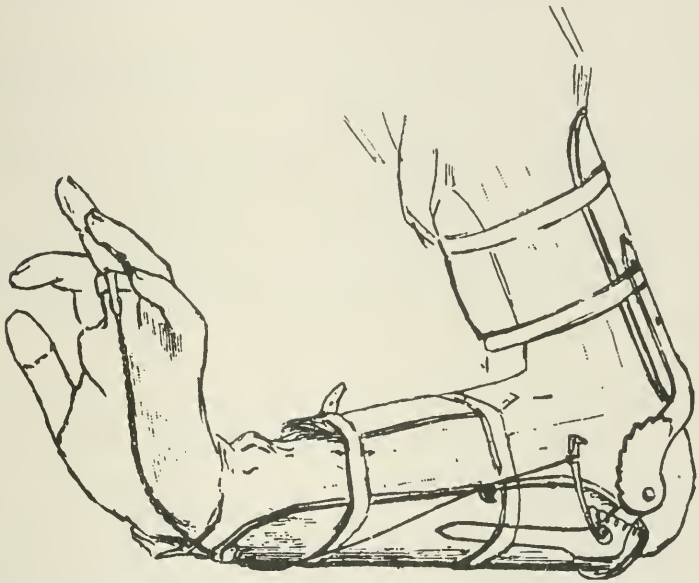


Fig. 51.—Position of disengagement. The catch lifted by hyper-extension of the hand becomes disengaged, and the forearm, being no more supported, falls automatically into extension on the arm. (Dagnan-Bouveret.)

attached to it a steel wire (fig. 50) which passes along the forearm-splint and, uniting with the wire from the other catch, is fixed to a small chain. This follows the palm of the hand, and is fastened to a ring which the patient wears on one of his fingers, preferably the index or the middle finger.

" When no traction is made on the chain, the catch bites on the serrated wheel, and the angle formed by the two splints cannot be increased ; it can only vary in the direction of greater flexion. If, on the other hand, the chain which passes to the catch is pulled,

the latter is raised and the angle formed by the two splints can be increased until they are eventually in a straight line with one another.

"The apparatus, which is worn on the shirt, is covered by the sleeve, so that the only part seen is the chain in the palm of the hand and the aluminium ring on one of the fingers.

"The working of the apparatus is very simple; by means of the sound hand or with the paralysed one, which many of these patients are able to make climb up their clothes, using pockets and buttonholes to help, the forearm is brought to the degree of flexion desired, and retains this position automatically.

"Then when the patient desires to extend his forearm he only has to hyperextend the hand, by which movement he pulls on the chain and disengages the catch, and the forearm falls down"[1] (fig. 50).

Appliances Suitable for Work in Patients Suffering from Injuries to Nerves

The course of paralysis following wounds is very slow; moreover, many cases will remain wholly or partially disabled. It is therefore important, from the social standpoint, to find some means of mitigating their disability.

It is most important that all neurologists and surgeons should use appliances which are suitable both for the nervous injuries presented by the patients and for the particular occupation in which they are engaged.

Obviously, even the best appliances will only mitigate the patient's disabilities, and in most cases the professional re-education of the wounded becomes an important question.

This problem is beginning to be partially solved

[1] Jean Dagnan-Bouveret, "Présentation d'un appareil de prothèse fonctionnelle pour les paralysies du plexus brachial supérieur," *Société de Neurologie*, January 6, 1916. *Revue Neurologique*, January 1916, p. 181.

both by the State and by private enterprise, but the neurologist must intervene in the choice of appliances intended to facilitate definite manual labour. A badly thought-out apparatus is not only inconvenient, but may even retard the progress of motor recovery. The following recommendation made by the Commission of the Neurological Society of Paris should therefore receive our warm approval:—

"Patients suffering from nerve injuries, in whose case a prosthetic appliance is indicated, should first be subjected to a special neurological examination, in which the conditions to be fulfilled by the appliance to be supplied to him must be specified.

"As soon as the patient has been given the appliance, he shall be examined again to make sure that it fulfils all the desired conditions."

CHAPTER IX

INTRANERVOUS LOCALISATION

The researches undertaken by Pierre Marie and Henry Meige in collaboration with A. Gosset[1] have clearly shown that the nerve fibres which go to different muscles are systematically arranged in the interior of the nerve-trunk, where they form bundles, each of which has its individuality.

In order to demonstrate this, the investigators had recourse to direct electrisation of nerve-trunks in operation wounds by means of an electric stimulator, which could be thoroughly sterilised, and which had been invented by Henry Meige (fig. 52).

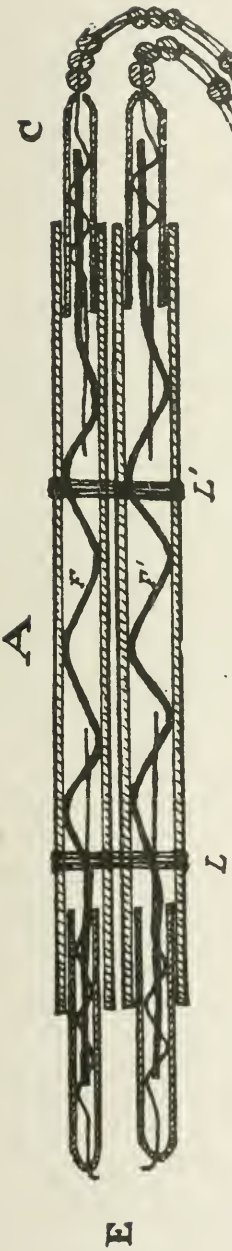
The investigations were made on injured nerves and neighbouring healthy nerves. The faradic current alone was used, its intensity varying in each case. For healthy nerves the coarse wire coil completely disengaged or even held at a distance from the primary coil gave a sufficient current to excite the nerve-trunks (in these cases it is even better to use a rheostat).

On the other hand, when the nerve was injured, a current furnished by the fine wire coil more or less engaged had to be used.

These investigations present many difficulties, which will necessitate subsequent researches in order to settle the question of some doubtful localisations.

First of all, if the nomenclature be used which is

[1] Pierre Marie, Henry Meige, and A. Gosset, "Les localisations motrices dans les nerfs périphériques," *Académie de Médecine*, December 28, 1915.



employed by anatomists to designate the different aspects of a nerve-trunk, it will be found that nerve-trunks are normally subjected to one or more torsions on their longitudinal axis, so that a certain amount of confusion may arise according to the level at which the nerve is examined. The difficulty is far greater in the case of an injured nerve incarcerated in a callus, or strangled by fibro-sclerotic proliferations. Its liberation, always an essential matter, occasionally necessitating chiselling the nerve out of the neighbouring cicatricial tissue, does not facilitate the investigation.

FIG. 52.—Henry Meige's electric stimulator.

A, *insulating apparatus*, consisting of two glass tubes connected by two ligatures, L L′. Into each of these tubes a bent ferro-nickel wire, F F′, is passed. E, *stimulating points*: two glass tubes entering the tubes of the insulator; into each tube a bent iron or platinum wire is introduced. The exciting end, whether pointed or blunt, projects 1 millimetre beyond the drawn-out end of the glass tube. The other extremity of the wire passes into the insulator and comes in contact with the ferro-nickel wire, which in turn penetrates into the small tube of the stimulating point and comes in contact with the nickel wire; by this device contact is doubly assured, as well as the rigidity of the apparatus. C, *conducting wires*: copper wires on which are strung fragments of glass tubes and an arrangement of interpenetrating wires similar to that described above (*Bulletin de l'Académie de Médecine*).

The following precautions in technique are necessary according to these writers in order to succeed in investigations of this kind. The nerve-trunk must be disengaged for a sufficient length (6 to 8 centimetres), so that it can be liberated from the neighbouring tissues.

After this freeing two sterilised and dried glass rods are slipped under the nerve-trunk, taking care not to allow the nerve to rotate, which would cause the guiding marks to be lost.

Care being taken to avoid injuring the nerve and changing its supports, the sterilised electrodes must be applied to that part of the nerve-trunk situated between the two glass rods, and then successively to all the points of its periphery.

The investigator must occupy himself exclusively with the exact localisation of the points to which he applies electrical stimulation.

Another observer records the resulting motor reaction.

A third observer varies the intensity of the electric current.

Each of them states his findings, which are noted for each fresh application of the electrodes.

At first a current of very feeble intensity should be used, which is progressively increased until a muscular response is definitely obtained.

If no movement occurs, one should make sure that the current is flowing well by applying the electrodes to another muscle in the wound which is not paralysed.

One must always be on one's guard against the phenomena of diffusion which occur when the current is too strong, or when the nerve has not been thoroughly isolated. Contraction will then be seen in the neighbourhood of the operation wound either in certain muscles which are innervated by other nerves than the one electrically stimulated, or in muscles supplied by the stimulated nerve, but innervated by filaments situated above the point of application of the electrodes.

All these difficulties which Pierre Marie and Henry Meige rightly emphasise show the complicated nature of an investigation of motor localisation, and how essential it is to make numerous examinations in order to be able to obtain absolutely certain results.

These authors investigated sixty-four cases, with the following results :—

Median Nerve

The motor bundles of this nerve appear to be arranged as follows in its course *in the arm.*

The fibres intended for the pronator muscles are situated in the *antero-external* region of the trunk of the median nerve.

The fibres for the *thenar* muscles are situated in the *posterior* region of the nerve.

With regard to the exact localisation of fibres intended for the *flexors of the carpus* and *flexors of the fingers* these investigations are less positive, since these muscles can be made to contract both by electrizing the internal as well as the external border of the nerve. Is this due to torsion caused by traumatism or surgical intervention, or is it due to individual peculiarities ?

However that may be, it can be taken that as a general rule the nerve fibres of the flexor muscles of the fingers predominate in the *postero-internal* region of the nerve.

When electrical stimulation is applied to the two heads of the nerve before their junction to form the V of the median a contraction of the pronators is obtained when the *outer head* of the nerve is stimulated, whereas stimulation of the *inner head* causes contraction of the *flexors of the fingers and carpus.*

Ulnar Nerve

The investigation of the localisation of the fibres in this nerve is a singularly laborious task, owing to its

smaller size and the difficulty in preserving its relations and identifying its various surfaces.

It is probable, however, that the nerve fibres for the flexor carpi ulnaris tend to group themselves towards the inner side, and the *fibres for the flexors of the fourth and fifth fingers* in the *postero-external* region.

The localisation of the fibres intended for the interossei, hypothenar, and adductor pollicis muscles is less definite.

According to clinical observations, however, it would appear that they occupy the anterior and internal aspect of the nerve.

Musculo-spiral Nerve

Electrization of the different surfaces of this nerve has demonstrated several localisations which appear to be constant. Especially is this the case with nerve fibres intended for the radial muscles which are situated in the *internal part* of the musculo-spiral in its course down the arm.

All the extensor muscles of the fingers appear to be under the control of fibres which occupy the postero-internal part of the nerve.

Lastly, the movement of supination is obtained by application of the current to the external region of the musculo-spiral.

Sciatic Nerve

In the case of the *internal popliteal*, fibres intended for the *gastrocnemius and soleus* are definitely localised in the *posterior* region of the nerve.

Those destined for the *tibialis posticus* are localised in the *antero-internal* region, and those intended for the *flexors of the toes* are situated in the postero-internal region.

In the case of the *external popliteal*, the fibres for the *peronei* occupy the *posterior* region of the nerve, those for the *tibialis anticus* occupy the antero-internal region, those for the *extensors of the toes* the antero-

external region, and those which are destined for the *extensor proprius hallucis* occupy mainly the *external* region.

These results should be compared, on the one hand, with those of M. and Mme. Dejerine[1] (confirmation by operation of the findings obtained by clinical examination), and, on the other hand, with the results obtained by the previous investigations of Stoffel.

With regard to the *musculo-spiral nerve*, M. and Mme. Dejerine and Mouzon adopt the following intra-truncular arrangement :—

Externally, the anterior cutaneous branch in front, then the branch to the supinator longus, and lastly the branches to the radial muscles further back.

Internally, the branches to the extensor carpi ulnaris and the muscles of the thumb in front, and then those to the extensor communis and that to the supinator brevis behind.

In the *ulnar nerve* the fibres are arranged thus in the region of the arm :—internally, superficially the cutaneous branches, those to the hypothenar eminence, then those to the interossei (those to the most external are placed furthest out), at the back the nerves for the adductor pollicis and flexor brevis pollicis. Externally, the nerves to the flexor carpi ulnaris and the flexor profundus.

In the *external popliteal* in the lower part of the popliteal region, the motor fibres are situated as follows :—internally, the tibialis anticus, next the extensor proprius hallucis, then the extensor communis; and externally, the peroneus longus and brevis.

In the *internal popliteal* the nerve bundles are situated as follows :—internally, the fibres of the external saphenous, then the plantar nerves, and the nerve to the inner head of the gastrocnemius; towards the external part, on the contrary, the nerves to the tibialis posticus, the flexor longus digitorum, the calcaneal branches, and the superficial branch of the external plantar.

[1] *Presse médicale*, No. 13, March 2, 1916.

We will now give a short account of the results obtained by Stoffel.

The musculo-spiral nerve above the bend of the elbow.

Anterior aspect.—Sensory branch.

External border.—Supinator longus.

Internal border.—Supinator brevis.

Posterior aspect.—Internally, the motor fasciculi of the common and special extensors, the abductor longus pollicis, and extensor carpi ulnaris. Externally, the extensors of the carpus.

The median nerve in the middle third of the arm.

Anterior aspect.—Sensory bundle and fibres of the thenar muscles (These two bundles together form three-fifths of the nerve according to Stoffel.)

External border.—Pronator muscles and flexors of the carpus.

Internal border.—Flexor sublimis digitorum.

Posterio-internal aspect.—The fibres of the flexor profundus, flexor longus pollicis, and pronator quadratus.

The ulnar nerve in the lower third of the arm.

The anterior aspect and the external border of the nerve are taken up by the large sensory bundle.

Internal border.—Flexor carpi ulnaris in front, and the flexor profundus digitorum behind.

The interossei, the adductor pollicis, and the hypothenar muscles are right at the back.

The *external popliteal nerve.*

In the trunk of the nerve itself, in the popliteal space, it will be found that the anterior tibial nerve is external; while the musculo-cutaneous, on the contrary, is postero-internal (see the internal popliteal).

The common trunk of the external saphenous nerve and the cutaneous peroneal branch is situated in the posterior and internal region of the nerve.

The internal popliteal nerve.

Anterior aspect.—Internally, the fibres for the flexor longus digitorum externally, those for the tibialis posticus.

External border.—Fasciculus for the flexor longus hallucis.

Posterior aspect.—Fasciculus for the gastrocnemius and soleus.

Examination of the trunk of the internal popliteal in the upper part of the thigh, where it forms the inner half of the great sciatic, shows that the nerve fibres of the posterior muscles of the thigh (biceps, semitendinosus, semimembranosus, adductor magnus) occupy the inner border of the internal popliteal.

From all these facts emerge some unquestionable conclusions which the surgeon should bear in mind as far as possible when performing a nerve suture.

In the case of the musculo-spiral nerve two fasciculi seem to be definitely established from a topographical point of view; *the fibres for the supinator longus occupy the external border of the musculo-spiral nerve* in the middle third of the arm, and *those for the extensor communis digitorum occupy the postero-internal region of the nerve.*

In the case of the median nerve during its course in the arm, *the fibres for the pronator muscles occupy the external border of the nerve,* and *those for the flexor sublimis digitorum occupy the internal border of the nerve.*

Although little definite appears to be known as yet of the localisation of the fasciculi for the thenar muscles, we may remind the reader of the circumstances mentioned above to which Stoffel attached great importance.

According to this writer, the fibres for the thenar muscles are mixed up with the sensory fasciculus of the median nerve, and this aggregation of sensory and thenar fibres takes up three-fifths of the nerve.

What we have said in dealing with surgical treatment concerning the numerous failures in sutures of the median and their determining causes shows the advantage in localising the different motor bundles so as to bring them to meet, as far as the exigencies of the operation allow, and to prevent the motor sheaths being invaded by the much more numerous sensory fibres.

Motor localisation is more uncertain in the **ulnar**

nerve than in any of the large mixed nerve-trunks of the limbs.

Our clinical examinations have, however, revealed that localisation on the internal border of the nerve of the *fibres for the interossei and the sensory fibrils of the nerve*, noted by M. and Mme. Dejerine and Mouzon, is very nearly correct, and that the localisation on the *external border of the ulnar nerve of the branches for the deep flexors of the last two fingers*, discovered by the method of Pierre Marie, Henry Meige, and Gosset, is correct and is confirmed by clinical experience.

In the **external popliteal** only one motor localisation appears to be constant, viz. that of the *fibres for the tibialis anticus on the antero-internal side of the nerve.*

Finally, in the case of the **internal popliteal nerve** the fibres destined *for the gastrocnemius and soleus occupy the posterior aspect of this nerve-trunk*, both in the region of the thigh and in the popliteal space, and *the fibres for the flexor longus digitorum the inner border of the nerve-trunk.*

An exact localisation of the motor fibres of the great sciatic and of its internal popliteal branch would be of great value for the same reasons we have detailed in the case of the median, as these reasons, in our opinion, partly explain the cause of failure in certain sutures of this nerve.

Systematically conducted investigations, thoroughly carried out on a large number of nerves, at different levels in their course, by different observers by means of the method of Pierre Marie, Henry Meige, and Gosset, may lead to definite conclusions.

These investigations should be made not only on injured nerves which are still excitable during an operation undertaken to free them, as is indicated in certain painful conditions, but also on healthy nerves, which can easily be exposed in operation wounds in the course of any of the operations which have to be performed for nervous, vascular, osseous, or muscular lesions.

We hasten to add that no harm has ever resulted from those investigations which we have seen carried out with all the requisite aseptic precautions and avoidance of any injury to the nerve-trunks, but they require a rapid and extremely careful operator of considerable experience.

CHAPTER X

PHYSIOPATHIC AFFECTIONS

Reflex Disorders, Congealed Hands, etc.

In the present chapter we propose to deal with a series of affections in which the main symptom is at one time a certain rigidity, at another time, on the contrary, a paresis, and sometimes, and more often, an association of rigidity and paresis, which is "neither true paralysis nor true contracture, but which results in almost complete immobilisation" (Henry Meige). These symptoms differ both from those seen in definite nerve lesions and in merely neuropathic (pithiatic) disorders.

The circumstances under which these conditions make their appearance, the thermal, trophic, and vascular symptoms which accompany them, and the stubbornness with which they resist all treatment, necessitate their being placed in a category of their own for the present. Their pathogeny and the problems raised as to their nature and the best method to adopt in dealing with them, both from a purely medical and military aspect, have given rise to large numbers of studies, of which we will endeavour to give a short account.

A. Symptomatology

These affections can be divided into three different categories, although this division is very schematic :—

1. Paralyses with contractures—the most typical cases.

2. Contractures.
3. Flaccid paralysis with hypotonus.

1. *Paralysis with Contractures*

Synonyms :—*Congealed hand* of Henry Meige, *complete paralysis of the hand* of Pitres, *acromyotonus* of Sicard, *reflex paralysis* of Babinski and Froment, *paratonic paresis* of Pierre Marie and Foix, etc.

This type of affection is chiefly seen in the upper limb, rarely in the lower limb.

It was in the year 1915 that the neurological centres first noted the appearance of certain stubbornly maintained attitudes of the limbs, and especially of the hand. They were accompanied by nearly complete functional impotence, which resisted all the physiotherapeutic methods which ordinarily improve rigidities or contractures resulting from osteo-articular or muscular lesions. Psychotherapy proved equally ineffective.

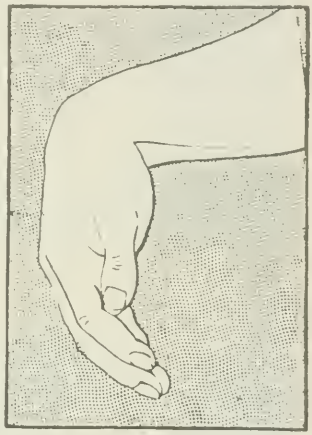


FIG. 53.—"Congealed hand."

At the Neurological Society of Paris, Henry Meige, Mlle. G. Lévy, and the present writer [1] drew attention to those disabilities following a wound of the forearm or of the hand, a wound which often left the nerves and bones intact, and which at times amounted merely to a simple contusion. H. Meige lays stress on the fact that the electrical reactions are but slightly affected, arterial pressure is equal to that of the sound side, and that frequently there is no impairment of the reflexes or sensibility, but that on the other

[1] Henry Meige, Mme. Ath.-Benisty, and Mlle. Lévy, "Impotence de tous les mouvements de la main et des doigts, avec intégrité des réactions électriques (main figée)," *Société de Neurologie*, November 4, 1915. *Revue Neurologique*, November–December 1915, pp. 1273–1276.

hand vasomotor and thermal disturbances are nearly always present. The various joints show a certain degree of rigidity, and after passive movement of the hand it returns to its congealed attitude, resembling the "accoucheur's hand" in its slightly dropped wrist, hollow palm, extended fingers, and in the thumb and little finger tending to approach the middle line below the three other fingers, which are themselves tightly applied to each other.

Babinski and Froment, who have devoted special attention to the study of this condition, have given a complete description of all the objective symptoms which differentiate it from known organic affections and from contractures or paralyses of an hysterical or pithiatic nature. The signs are as follows: [1]—

(*a*) The presence of very marked *vasomotor disturbances*, generally with segmentary distribution, which does not correspond to a definite nerve area and predominates at the extremities.

The "congealed hand" or the paretic foot have generally a cyanotic or salmon-pink appearance. The

[1] J. Babinski and J. Froment, "Les modifications des réflexes tendineux pendant le sommeil chloroformique et leur valeur en sémiologie," *Académie de Médecine*, October 19, 1915.

J. Babinski and J. Froment, "Sur une forme de contracture organique d'origine périphérique et sans exagération des réflexes," *Société de Neurologie*, November 4, 1915.

J. Babinski and J. Froment, "Contribution à l'étude des troubles nerveux d'origine réflexe. Examen pendant l'anesthésie chloroformique," *Société de Neurologie*, November 4, 1915.

J. Babinski and J. Froment, "Paralysie et hypotonie réflexes avec surexcitabilité mécanique, voltaïque et faradique des muscles," *Académie de Médecine*, January 11, 1916.

J. Babinski and J. Froment, "Contractures et paralysies traumatiques d'ordre réflexe," *Presse médicale*, February 24, 1916.

J. Babinski and J. Froment, "Des troubles vasomoteurs et thermiques d'ordre réflexe," *Société de Neuroloique*, March 2, 1916.

J. Babinski, *Les caractères des troubles moteurs dits "fonctionnels" et la conduite à tenir à leur égard.*

Rapport à la *Société de Neurologie*, meeting of April 6–7, 1916. *Revue Neurologique*, April–May 1916.

J. Babinski, J. Froment, and Heitz, "Des troubles vasomoteurs et thermiques dans les paralysies et contractures d'ordre réflexe," *Annales de Médecine*, September–October 1916.

PLATE III.

Two varieties of " congealed hand " compared with the sound hand.

slightest pressure on the skin causes a blanched area which does not disappear for some time.

(*b*) *Local hypothermia* is permanent and often pronounced. Measurements with a thermo-electric apparatus show that there may be differences of 4° to 5°, or even 8° C., between the affected and healthy side.

(*c*) The *systolic pressure* is equal on both sides, but *the amplitude of the oscillations* is always slighter on the affected side. After chilling the limbs, this difference may become considerable. It grows less marked, disappears, or becomes inverted under the action of heat.

On the affected side Babinski found a diminution of hæmoglobin and the red corpuscles on examination of the blood by the colorimetric method. By the spectroscopic method the hæmoglobin appeared less reduced on that side.

(*d*) *Atrophy of all the tissues* of the hand and fingers, with occasional increase in size of the joints, resembling those of patients affected with chronic rheumatism.

(*e*) *Moisture* and occasionally *maceration* of the skin.

(*f*) *Decalcification* of the skeleton of the affected limb, clearly recognisable in the skiagram.

(*g*) *Hypotonus*, especially marked in the purely paralytic forms, but seen also in certain cases showing contractures.

(*h*) *Mechanical hyperexcitability of the muscles* of the affected limb, especially noticeable in the paretic type, but also seen in the type accompanied by hypertonus.

This hyperexcitability of the muscles is most obvious in the small muscles of the hand or foot (thenar, hypothenar, interossei, extensor brevis digitorum, and muscles of the sole of the foot). Even slight percussion of the muscles on the affected side elicits a slow movement of great amplitude.

(*i*) *Modifications in electrical contractility.* These are purely quantitative and are not accompanied by any signs of R. D., and consist simply in *faradic and*

voltaic hyperexcitability, premature fusion of the contractions, and more rarely slight *subexcitability*.

This hyperexcitability to mechanical and electrical stimuli may also be seen in the nerve-trunks. But both in the nerves and in the muscles it varies with the temperature. If both limbs are artificially refrigerated it becomes accentuated on the affected side, while if, on the contrary, the affected limb is heated, muscular superexcitability becomes less.

(*j*) The *tendon reflexes* are but slightly modified.

(*k*) If the patient is put under *chloroform narcosis*, one finds that the contracture often does not disappear until deep anæsthesia has been reached, and that it returns at the same time as the tendon reflexes, sometimes before the reappearance of the conjunctival reflex. In addition during chloroform narcosis the *reflexes of the affected limb become exaggerated*, and for a very long time clonus of the injured side can be noticed when the reflexes of the healthy limbs are already abolished.

Pierre Marie and Foix[1] added several remarks to this symptomatological complex. According to them the "congealed hand" is kept flexed on the forearm by a contraction of the palmar muscles, which can be seen standing out under the skin.

The condition considered as a whole constitutes a *paretic state*, with weakness of all movements and apparent inability to execute some of them.

The movements which are most impaired according to Pierre Marie and Foix are those of abduction and extension of the thumb. During slight movements of the hand and in a state of repose one can recognise very clearly the *hypertonus* of some of the muscles (flexor carpi radialis, palmaris longus, interossei and adductor pollicis), and the *hypotonus* of the others (muscles of the lower area of the musculo-spiral). When the radial muscles contract there occurs at the

[1] Pierre Marie and Foix, "Sur une forme spéciale de 'parésie paratonique des muscles moteurs de la main,'" *Société Médicale des Hôpitaux*, February 4, 1916.

same time a contraction of the palmar muscles, which are in a state of hypertonus, and this contraction opposes the efficacy of the action of the radial muscles.

Muscular hyperexcitability to mechanical stimuli is said to predominate in the case of muscles supplied by the median and ulnar (thenar muscles, flexors, interossei and hypothenar muscles).

Unipolar faradic stimulation of the back of the forearm causes flexion of the hand instead of the normal extension. This fact would indicate a weakness of the extensor muscles of the hand, as has been pointed out by Babinski.

The trophic disturbance finds expression in the diminution of size and flabbiness of the hand, the thinning of the skin, which becomes finer and often of a pink tinge, and in the occasional deformity and striation of the nails.

2. *Contractures*

There is not always a definite line of demarcation between the conditions just described and pure contractures; generally a certain degree of paresis of some muscles is associated with contracture of others.

Contractures of the upper limb generally predominate in the hand and in the fingers; the biceps is fairly frequently affected.

According to André Léri and Edouard Roger the most frequent varieties are extension contracture of the hand and fingers (fingers extended or hyperextended either all together or in groups of two or three, and separated from one another). A more frequent variety is the classical attitude known as "accoucheur's hand," with the fingers extended and brought close together and pressed against each other.[1]

[1] André Léri and Edouard Roger, "Sur quelques variétés de contractures post-traumatiques et sur leur traitement," *Société Médicale des Hôpitaux*, October 22, 1915.

According to Sicard,[1] contractures of the hand and fingers adopt certain attitudes which can be schematically classified under the following five headings:—

(*a*) "*Fist*" appearance. All the fingers are powerfully bent into the palm of the hand. This is due, according to Sicard, to contracture of the flexors of the forearm ("median nerve hand").

(*b*) "*Holy water basin hand.*" The palm of the hand is hollow, and is surrounded by the half-bent fingers, due to contracture of the interossei and the adductor pollicis ("ulnar nerve hand").

(*c*) "*Spindle hand,*" due to contracture of the extensors of the hand, fingers, and interossei ("musculospiral and ulnar nerve hand").

(*d*) "*Swan-neck hand,*" or "median and ulnar nerve hand," flexed at an acute angle at the wrist with the fingers extended.

(*e*) "*Sign-post hand*" (flexion contracture of the last two fingers, and extension of the index).

All these contractures can easily be distinguished from fibro-tendinous retractions by applying Esmarch's rubber band to the root of the limb as far as possible away from the group of muscles which has undergone contracture (Sicard). Circulation in the limb is thus suppressed, and after several minutes the contracture vanishes. In retraction, on the other hand, the position of the limb does not alter. The efficacy of this method can be increased, according to Sicard, by injecting locally in the neighbourhood of the muscular tendons involved a few cubic centimetres of a 1 per cent. solution of stovaine and cocaine. As soon as the elastic band is relaxed, contracture of the parts reappears as pronounced as ever.

Sicard adds the following features which characterise the contractures with or without paresis, which he has denominated *acromyotonus*:—

[1] J. A. Sicard and Imbert, "La bande de caoutchouc et l'alcoolisation locale des nerfs dans le traitement des contractures par blessures de guerre," *Société Médicale des Hôpitaux*, April 16, 1915.

J. A. Sicard, "L'alcoolisation tronculaire au cours des acromyotonies rebelles du membre supérieur," *Paris Médical*, June 3, 1916, No. 23.

A

B

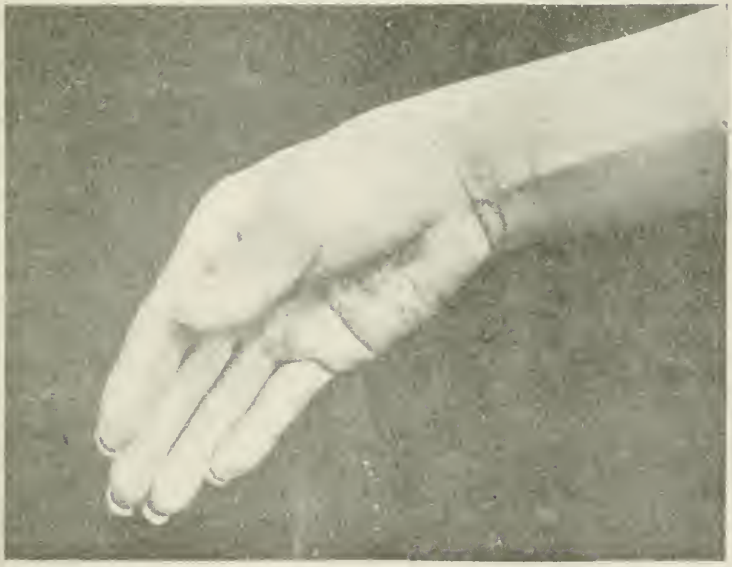


PLATE IV.

"Congealed hand."

A, profile view. B, palmar view.

"Their appearance after wounds of connective tissue, muscle, or bone, quite independent as a rule of any direct injury to a nerve-trunk.

"Their extension to neighbouring muscular areas quite independent of the sphere of injury.

"Their frequent localisation in the extremities, and especially in the hand and fingers.

"Their painless character so long as the hypertonic position is not interfered with, and the paroxysms of pain when any correction is attempted.

"Their aggravation by the local application of massage or electricity or by direct mobilisation.

"Their disappearance under general anæsthesia and a rubber band; their immediate reappearance on the cessation of general anæsthesia or local ischæmia.

"Their partial and transitory improvement as the result of very gentle manipulations.

"The characteristic, persistent, and penetrating odour of maceration which is occasionally given off by the hand and fingers which have undergone contracture.

"Their resistance to ordinary treatment and even to prolonged extension in plaster apparatus" (Sicard).[1]

In some of these cases, according to Sicard, a marked exaggeration of the bone reflexes may also be found, as is shown, for example, by generalised flexion of the fingers and hand on percussion of the metacarpal bones, etc.

Other types of contractures of the upper limb have been described by Babinski and Froment, *e.g.* the upper limb extended and tightly pressed against the body; the upper limb folded on itself and describing a Z, with the forearm bent and the wrist dropped, etc.

Contractures of the Lower Limb.—These can affect the extremity of the limb just as in the case of the upper limb, but they frequently also involve the knee and the hip.

In the foot, the following attitudes may be seen: talipes varus and talipes equino-varus and extension

[1] *Paris Médical*, June 3, 1916, pp. 510–511.

contracture of the last four toes with flaccid paralysis of the big toe (Babinski and Froment).

The contractures of the leg may be either in flexion or extension.

In the last case they are often associated with talipes varus.

In the hip contractures of several of the muscles of the pelvic girdle may be observed, often involving external rotation of the lower limb. Frequently these contractures of the hip muscles are accompanied by paresis and sensory disturbances in the foot, and by the abolition of the plantar cutaneous reflex (Babinski and Froment). Artificial heating of the foot may cause this reflex to reappear.

Symptoms of paresis, as we have seen, are very often associated with the usual signs of contractures. Their objective characteristics are exactly the same as those found in affections of the first category, viz. vasomotor troubles, hypothermia, atrophy of the entire limb, decalcification of the skeleton, and hyperexcitability of the muscles to mechanical stimuli and faradic and galvanic currents.

It was among wounded men affected by contractures of the lower limbs that Babinski and Froment conducted their very interesting investigations on the modification of the tendon and osteoperiosteal reflexes under the influence of chloroform narcosis.

While the tendon reflexes (knee-jerks, ankle-jerks) are in some cases definitely exaggerated when compared with the healthy side, in other cases they are hardly modified at all, but under general anæsthesia the difference between the healthy and the affected side is marked and indicates a hyperexcitability of the spinal centres (Babinski and Froment).

It is often possible in these cases to find patellar clonus on the affected side even when all the other reflexes are abolished. In like manner the knee- and ankle-jerks reappear first during the period of recovery from the anæsthetic, and are far more marked than those of the sound side.

The determining causes of contractures of the lower limb are also relatively slight, such as seton-like wounds of the soft parts of the leg or thigh, superficial wounds of the foot, and simple contusion of the hip or knee.

3. *Flaccid Paralyses with Hypotonus*

They are less frequently seen in a pure condition than the varieties just described, and are often associated with more or less marked contractures.

They are frequently situated in the upper limb, and affect by preference the hand which shows wrist-drop, together with a much more marked hypotonus of the extensors of the carpus and the hand than is seen in paralysis following a complete division of the musculo-spiral nerve.

Contracture of the biceps may coincide with the wrist-drop.

In all these affections the phenomena are constantly found to which Babinski and Froment have drawn attention, especially hypothermia and the hyperexcitability of muscles to mechanical stimuli.

B. Pathogeny

The pathogeny of these affections has been very much discussed.

Babinski and Froment,[1] André Léri and Edouard Roger,[2] and Guillain and A. Barré [3] were the first to draw attention to the necessity of distinguishing these disorders from manifestations of pithiatism or simulation.

[1] Babinski and Froment, " Les modifications des réflexes tendineux pendant le sommeil chloroformique et leur valeur en sémiologie," *Académie de Médecine*, October 19, 1915.

[2] André Léri and Edouard Roger, " Sur quelques variétés de contractures post-traumatiques et sur leur traitement," *Société Médicale des Hôpitaux*, October 22, 1915.

[3] Georges Guillain and A. Barré, " Les contractures dans la pathologie nerveuse de guerre," *Bulletin de la Société Médicale des Hôpitaux*, January 21, 1916.

The interpretation of the pathogeny differs, however, with each writer.

Babinski and Froment regard these lesions as being of a *reflex* character analogous to the disorders noted by John Hunter in 1839, and studied under the name of *reflex amyotrophies* by Vulpian and Charcot. The last observer interpreted them as the manifestation of a condition either of stupor or of exaltation of the cells of the corresponding spinal centres.

Babinski and Froment consider the hypothesis of Charcot as possible, but incline to the idea that owing to the importance of the vasomotor and thermal disorders which influence the condition of the muscles, it might be possible to assign the sympathetic system an important share in the genesis of these conditions.

In point of fact, the mechanical hyperexcitability of muscles and nerves seems to be closely related to hypothermia.

Refrigeration increases muscular excitability on the affected side, especially in the small muscles of the extremities.

After immersion in hot water, on the other hand, muscular contraction becomes rapid and less intense.

Premature fusion of the contractions disappears also.

Under the influence of heat, the appearance of tremors and an appreciable exaggeration of the tendon reflexes may occasionally be observed.

Babinski and Froment suppose that these phenomena are due to a vaso-constrictor spasm produced by a reflex irritation originating in a peripheral lesion.

On physiological grounds, these writers hold that "side by side with the condition of anæmia and hypothermia a third factor inevitably comes into play, when the action of these two is prolonged, viz. the accumulation of physiological poisons, which deserves careful attention. The muscle then behaves as in the first stages of veratrine poisoning." [1]

[1] J. Babinski and Froment, "Des troubles vasomoteurs et thermiques d'ordre réflexe," *Société de Neurologie*, March 2, 1916. *Revue Neurologique*, March 1916, p. 410.

Babinski and Froment remark that the disorders in question are chiefly manifested by physiological anomalies, and have therefore suggested that they should be known under the name of *physiopathic disorders*, which is a non-committal term as regards their pathogeny, and enables them to be distinguished from affections of which the organic origin is known.

Léri and Edouard Roger, while guarded as to the origin of some of these contractures, which they consider as purely functional and hysterical, draw attention to a number of genuine cases which give rise to these contractures.

The first category of muscular contractures is due to an irritation of the muscle by *metal dust* in the fleshy part of the muscle, as is shown by radiography. These writers are inclined to think that other foreign bodies may sometimes be incriminated, which are neither metals nor bones, and are invisible on the screen, but are nevertheless able to determine powerful contractures of the biceps or the flexor muscles of the leg, less by irritation of the motor filaments than by direct irritation of the muscle fibres themselves. On other occasions they regard this irritation of the muscle fibres as caused by a very adherent cicatrix, especially when suppuration has been prolonged or when the path of the projectile has been in the neighbourhood of a tendon.

In a second and rarer group, according to these authors, the irritation does not affect the muscle but the nerve itself. These contractures may be confined to a single muscle or a single group of muscles, which is logical, seeing the specialisation of nerve fibres in the interior of the nerve-trunks.

Finally, a third variety of organic contractures can occur in the *muscles* which are *antagonistic* to the *paralysed muscles*. Thus there are musculo-spiral paralyses, in which the wrist, instead of dropping, rises and becomes rigid owing to violent contracture of the flexors of the fingers and carpus.

According to Georges Guillain and A. Barré, we must distinguish :—

1. Contractures due to compression, irritation of the *nerve*, a foreign body (bony or metallic), by cicatricial fibrous tissue, to incomplete wounds of a nerve with or without painful neuroma affecting by reflex action the adjacent spinal centres, the reaction of the nerve in this case being often manifested by contracture and not by paralysis.

2. Contractures due to irritation of the *muscle* by a foreign body (metal dust, callus, etc.).

3. Contractures caused by ischæmic constriction[1] due to an appliance put on too tightly after injury. Besides this pure ischæmia we must also take into consideration compression of nerve-trunks and muscles.

In a subsequent study Guillain and Barré[2] record a certain number of contractures with paresis due to an *irradiating neuritis.*

In support of this pathogeny they refer to the exaggeration and diffusibility of the reflexes, and the existence of pains arising both spontaneously and on pressure of the nerve-trunks situated above and below the wound.

The explanation of these objective modifications is to be found, according to Guillain, " in the rôle played by the nerves in the conduction of microbes and toxines and that of ascending neuritis, the latter being considered rather from the physiological than from the anatomical standpoint." [3]

The rôle of the sympathetic and the factors of immobilisation and constriction of the limb must also be taken into consideration.

" The many poisons—microbial and cellular—causing hæmolysis and cytolysis may cause the dis-

[1] G. Guillain et A. Barré, " Les contractures ischémiques," *Réunion Médicale de la VI^e Armée*, January 12, 1916.

[2] Georges Guillain and A. Barré, " La névrite irradiante," *Société Médicale des Hôpitaux*, April 7, 1916.

[3] G. Guillain, " Les névrites irradiantes et les contractures et paralysies traumatiques d'ordre réflexe," *Société Médicale des Hôpitaux*, May 26, 1916.

tant secondary affections, of which we see the clinical manifestations, either by the lymphatic channels of the nerves or by the nerve-trunks themselves" (Guillain).

Some authors are inclined to think that all the unquestionably organic symptoms of these affections may be possibly due in the first place to *prolonged immobilisation of the limb*. This immobilisation is necessary at first for the treatment of the wound, and is then due to the employment of various appliances (splints, plaster of Paris, etc.) used for correcting the vicious position of the limb, and is continued subsequently by the patient consciously or unconsciously.

This hypothesis gave rise to a long and important discussion at the meeting of the Neurological Society of Paris on 6th and 7th April 1916.[1]

According to Henri Claude, these cases occur, as a rule, among wounded men who have been sent to units where their wound received the chief attention without any movement of their joints, from fear of causing them pain. "They were put in splints for weeks or months; when the dressings were removed, ill-advised suggestions made them think that the use of their limb was impaired, and the medical officer was afraid of causing them suffering by rectifying the vicious attitude of their limb, which became more and more exaggerated in consequence. . . .

"If the patient is left to himself and wrongly advised, or if he present that apathetic and indifferent frame of mind seen in so many patients for whom hospital life is almost a social condition, the abnormal attitude which primarily indicated a reaction of defence or a painful reflex is rendered permanent by a psychical process of an hysterical nature, or simply by habit. . . .

"Whatever the mechanism of the *fixation of the vicious attitude* may be, it gets more and more exaggerated as time goes on, and becomes less and less

[1] *Revue Neurologique*, April–May 1916, pp. 524–572.

reducible, owing to the articular and muscular lesions which are often present from the beginning of this affection, but become progressively aggravated in all cases" (Henri Claude).[1]

Jean Camus thinks that in a fairly large number of these affections "prolonged immobilisation of the limbs is the only explanation acceptable of these late symptoms.

"Immobilisation is due to many causes, such as too long application of a plaster apparatus, or large and irregular wounds with fistulous openings which have taken months to cicatrise; in other cases immobilisation has either been the wish of the patient from the first, or, what is more frequent, has been adopted after immobilisation originally due to an apparatus or a dressing.

"In these cases a reflex does not give rise to the symptoms, but the cause resides in the wish of the patient to become immobilised or to persist in an immobilised condition which he did not create himself in the first place." [2]

André Thomas is inclined to think that the thermal and circulatory disorders are the result of prolonged immobilisation and inertia.

"The wasting of the limb and decalcification of the bones may, in a very large proportion of cases, be the result of immobilisation or inertia, but it is not improbable that in some cases the muscular atrophy may be due to the same cause as the so-called reflex muscular atrophies." [3]

Henry Meige and A. Thomas have also emphasised the fact that reflex contractures and paralyses are very rare in severely wounded cases whose disabilities are incurable.

According to Tinel, the organic factor in these affections is nearly always a slight nervous lesion of the nature of neuritis, but this idea of neuritis does

[1] *Revue Neurologique*, April–May 1916, pp. 532–536.
[2] Jean Camus, *Revue Neurologique*, April–May 1916, pp. 540–542.
[3] *Revue Neurologique*, April–May 1916, pp. 542–546.

not seem sufficient to him to explain the intensity of these contractures, their persistence, and their *progressive aggravation.*

"An important functional factor must certainly be adduced in addition, undoubtedly more important as a rule than the actual neuritis, viz. *muscular inaction*, *prolonged immobilisation*, and *moral inertia*, which almost all these patients display.

"There is therefore a problem to solve: the symptoms found have exactly the appearance of organic troubles; the circumstances of their appearance, on the other hand, have something perplexing, illogical, and paradoxical, which makes them more allied to functional and hysterical disorders" (Tinel).

Babinski and Froment[1] protest against the importance attributed to immobilisation. They think, on the contrary, that these disorders are likely to develop without there having been any immobilisation, properly speaking, at any time.[2]

For our part, we think that a critical examination should be made of the facts which have been reported and variously interpreted.

First of all, one point should be noted; that is, that war wounds can determine contractures and flaccid pareses with hypotonus.

Thus we had the opportunity of seeing a very marked and irreducible contracture of the biceps in the course of complete section of the musculo-spiral nerve in the middle third of the arm, without fracture of the humerus. This contracture came on immediately after the wound; besides which the body of the muscle itself appeared of harder consistence than that of the opposite side, and there existed at this level an obvious hyperexcitability to mechanical and electrical stimulation when compared with the healthy side.

On many occasions we have been able to observe

[1] *Revue Neurologique*, April–May 1916, pp. 546–549.

[2] Babinski and Froment, "Troubles nerveux d'ordre réflexe ou syndrôme d'immobilisation," *Société de Neurologie*, May 4, 1916. *Revue Neurologique*, July 1916, p. 914.

these contractures of the biceps, and eventually a certain degree of tendinous retraction supervened.

What is the determining cause of the contracture in these cases ?

Is it a lesion of the muscle itself due to metallic dust, a fibrous cicatrix or other foreign bodies invisible on radiography, as Léri affirms, or is there an irritation of certain motor or sensory nerve fibres constituting that ascending contracture of which Ducosté speaks,[1] or should it be attributed to a lesion of the periosteum or of the articular fibrils of the neighbouring joint ?

We are unable to say. We will confine ourselves to reporting other similar cases.

A patient suffering from a wound of the lower jaw, with injury of the cervico-facial branch of the facial nerve, showed a very obvious contracture of the clavicular head of the sterno-cleido-mastoid. This contracture did not inconvenience him in any way, he did not complain about it, and it was only revealed by medical examination.

We had the opportunity of seeing other partial and transitory contractures in the course of lesions of the brachial plexus, accompanied by irritation of the spinal cord,[2] contractures of the trapezius on the healthy side, or of the pectoralis major on the paralysed side, and of certain nuchal muscles. This last variety was characterised by pain and aching in the contractured muscle, which was hard and hyperexcitable on percussion with the hammer or by electric currents.

We also saw an example of flaccid paresis with marked hypotonus of the whole right upper limb.

The wound (a grazing of the muscles of the anterior aspect of the forearm) had not involved any important nerve. The patient, however, showed a complete atrophy of the whole limb and the muscles of the shoulder girdle, with very marked flaccidity and

[1] Maurice Ducosté, " Les contractures dans les lésions nerveuses périphériques," *Société de Biologie*, July 24, 1915.

[2] Mme. Athanassio-Benisty, *Formes cliniques des lésions des nerfs.* Masson, 1916, pp. 158–159.

incomplete and feeble movements. Movements of the flexors were limited by a kind of stiffness of the fingers. There was slight hypo-excitability to electrical stimulation, but the strongest sign in favour of an organic lesion was the complete abolition of all the tendon and periosteal reflexes of the upper limb, including the scapular reflex.

The patient had been buried by the explosion of a mine, and the act of rescuing him had doubtless been the cause of pulling on the cervical roots, possibly giving rise to an ascending lesion, which had reached as far as the cells of the anterior group of the cord.

We may thus find in the traumatic neuropathology of war, contractures as well as flaccid paralyses even without direct injury to the nerve-trunk. A reflex mechanism can certainly be invoked to explain these phenomena. We confine ourselves for the present to reporting these facts, without suggesting an interpretation.

The affections described above, in which the symptomatology appears well established, but in which the pathogeny is still under discussion, can, in our opinion, be classified in four groups according to their determining causes :—

1. Painful neuritis.
2. Direct involvement of the muscles.
3. Involvement of the bones, fibrous tissue, and tendons.

These three groups must be carefully distinguished from a fourth group, which comprises another form of contractures and hypotonic conditions, viz. those in which the initial cause is only a slight wound (seton-like wound, contusion) and in which the subsequent symptoms mainly consist of a prolonged immobilisation.

1st Group.—*Painful neuritis.*—We have here to deal with slight wounds of nerves, chiefly involving the median or ulnar in the forearm and wrist (more rarely in the arm), and constituting a painful form of neuritis, as is proved by a frequent history of definite causalgia,

presence of pain on palpation of the nerve-trunk, articular rigidity, vasomotor and secretory disorders, generalised atrophy, and finally a more or less appreciable degree of decalcification of the skeleton.

2nd Group.— *Various muscular lesions—Constriction —Lesions of sensory nerve fibres.*—This group comprises contractures due to wounds of muscles or to constriction (plaster appliances). Possibly they may occasionally be due to an irritation of the peripheral sensory fibres determining, by a reflex mechanism in the spinal cord, an increased nervous influx to certain muscles according to a radicular or peripheral distribution.

In any case, the possibility of direct irritation of the muscular fibres must not be lost sight of; thus we have seen a case of persistent contracture of the triceps due to the inclusion of a piece of a coat in a wound of the elbow. When this foreign body was removed the contracture disappeared.

3rd Group.— *Lesions of the bones, muscles, and tendons.* —In this group we have to deal with injuries to the muscles or tendons, involving the periosteum, or even fracture of the neighbouring bones, which have suppurated for long periods, and necessitated the use of plaster apparatus, or at least prolonged immobilisation in a sling.

We shall see in a later chapter how prolonged suppuration affects the joints of the extremities of a limb, especially the hand. These joints become inflamed, and the inflammation results in very obstinate rigidity of the affected joint.

Suppuration in bones also gives rise occasionally to considerable decalcification of the hand and fingers, a thinning of all the integuments, and a general wasting of the muscles similar to that seen when the intra-nervous sympathetic fibres are subjected to irritation.

4th Group.—*Congealed hand.*—Finally, a fourth group comprises contractures of the nature of club-foot and that form of paralysis combined with hyper-

tonus of the hand, which is best expressed by the name of *congealed hand.*

In the first place, there may be only a simple contusion or trifling wound, or, on the other hand, seton-like wounds of the soft parts of the forearm, leg, or thigh, and more or less transfixion of the extremities, but with only slight injuries to bones or tendons.

It is principally in these cases that an important pathogenic rôle can be attributed to *immobilisation.*

The limb has first been immobilised on account of pain and suppuration. The resulting stiffness has not been treated by massage and other physiotherapeutic methods, and the part has been confined in plaster or in splints.

These means of restraint have increased the rigidity, and their pressure, which at times has been very marked, has caused atrophy, and then a paresis of certain muscles, especially of those supplied by the musculo-spiral, the tonus of which muscles is inferior to that of the flexors. A certain hypertonus, and contracture of some other normally very powerful muscles, such as the flexors of the carpus and the fingers, the adductor pollicis, the interossei, etc., then appear, and give rise to vicious positions.

After giving up these appliances, the patient continues to immobilise the affected limb consciously or unconsciously.

The articular and muscular disorders may also become aggravated by the immobilisation and chilling which result from it, and by all the modifications which these different factors bring about in the structure of the muscles.

It is therefore not unlikely that immobilisation enters to a certain extent into the etiology of "congealed hand." This hypothesis does not in any way exclude that of the reflex mechanism suggested by Babinski and Froment.

In addition, it may also be advanced, as has already been done by the present writer in conjunction with Henry Meige, and as Babinski and Froment incline to

believe, that in these reflex phenomena not only are the spinal nerve paths called into play, but the sympathetic as well. The thermal, vasomotor, and secretory disorders, which are rarely absent in these cases, support this view.

Whatever the pathogeny of this condition may be, the reality of these phenomena is incontestable, and we actually find ourselves in the presence of a group of clinical facts which it is essential to differentiate.

One more fact deserves to be mentioned, on which Henry Meige has laid special stress.

This is the peculiar mentality of the majority of these patients, especially those who have "congealed hands."

"A peculiar state of mind, consisting in torpor and inertia, combined with the constant obsession of their infirmity; they keep their eyes fixed upon it, and protect it with the other hand; they gloat upon it, so to speak, but yet they are in no pain. This solicitude, associated with their passive inertia, and the absence of all organic symptoms, and often even of any apparent wound, has not infrequently made one think that such cases are exaggerators or simulators" (Meige).[1]

"Solitary, anxious, and careworn, making no unnecessary gesture, and perpetually on the lookout, they show a perfect motor negativism for the affected limb.[2]

"It seems sometimes as if one were in the presence of one of those localised *motor amnesias* which give rise to a condition of *aboulia* for certain movements, and which are undoubtedly of psychopathic origin."

It is important to take into account this mental factor, which, even if it does not of itself constitute the whole affection, undoubtedly aggravates the motor disturbance.

In the lower limb this immobilisation and the retention of vicious attitudes often result in contrac-

[1] *Revue Neurologique*, April–May 1916, pp. 550–551.
[2] *Ibid.*, November–December 1915, p. 1276.

tures and paresis, shown by various disturbances of gait.

Limping following commotion, contusions of the hip or knee, or superficial wounds is often justified at first by more or less severe articular or peri-articular lesions, but is persisted in as a bad habit.

Henry Meige, who has made a study of these forms of limping, sums up their clinical history in the following formula: "Habit creates the aptitude for the attitude." [1]

The majority of these contractures, which are limited to one group of muscles of the thigh, are not irreducible, as can be found by using the *diplocinetic method* advocated by H. Meige:

"Vigorous traction or pressure is *simultaneously* exerted on the different segments of *both* lower *limbs* whilst *rapidly* giving successive commands, such as 'straighten,' 'bend,' 'pull,' 'push,' 'separate,' 'bring together,' etc., so quickly that the patient has not time to prepare his muscular answer, but obeys mechanically."

This method, well followed out, enables one to judge up to what point the rigidity or disappearance of voluntary motility is real or assumed.

C. Treatment

All authors agree that these affections are obstinate, and that treatment such as is usually prescribed generally ends in failure.

Psychotherapeutic measures, counter-suggestion in particular, do not affect the progress of this condition, and, according to Babinski and Froment, this is the principal characteristic which distinguishes these disorders from pithiatic manifestations.

The physiotherapeutic measures which can be used are but few. Recourse must be had to very gentle

[1] Henry Meige, "De certaines boiteries observées chez les 'blessés nerveux,'" *Société de Neurologie*, October 7, 1915. *Revue Neurologique*, November–December 1915, pp. 939–947.

manipulations in order not to cause pain and increase stiffness and contracture. The same applies to electrization, and especially to intense faradic currents.

Immobilisation in plaster is always bad, and only serves to increase the vicious attitude.

Diathermy sometimes gives good results according to Babinski.

A large number of writers nevertheless think that pyschotherapeutic measures are essential. Thus Grasset proposes semi-isolation of these patients in a special hospital, forbidding them to go into the town except on a Sunday with the special permission of the medical offcer.

Professional and *military re-education* should not be neglected. In short, all that we have said previously about the obscure pathogeny of these affections indicates that treatment must vary with each case.

In lesions due to injuries of the nerves, muscles, or bones, gentle but persevering mobilisation will prevent irreducible ankylosis.

In the case of " congealed hands," or contractures following slight wounds or contusions, early and energetic mobilisation, in combination with firm psychotherapeutic measures, employed *in units at the front*, will soon put an end to commencing rigidity. Sick leave must be refused, as it nearly always has a bad effect in these cases.

In obstinate forms in which treatment does not cause any obvious improvement it is better to discharge the patient temporarily or put him under medical supervision, since in the absence of such supervision the condition always tends to remain stationary.

It is for those stubborn contractures, causing complete loss of power in the hand and fingers, which cannot be improved by any of the usual treatments, that surgical interference has been suggested.

J. Sicard [1] has proposed alcohol injections into the

[1] J. H. Sicard, " L'alcoolisation tronculaire au cours des acromyotonies rebelles du membre supérieur," *Paris Médical*, June 3, 1916.

nerve supplying the muscles suffering contracture. By this process "relaxation of the contracture and disappearance of the pain for a certain time is obtained."

The technique is as follows:—First of all a skiagram is taken of the limb to see if there is any foreign body present; if there is, this must be extracted. After local anæsthesia the nerve-trunk is exposed and anæsthetised with 2 cubic centimetres of a 1 per cent. solution of cocaine, and about 1 cubic centimetre of alcohol at 20° is injected into the nerve-trunk.

"Almost immediately after the injection, sometimes only after the lapse of several minutes, muscular relaxation occurs, and cutaneous anæsthesia appears in the injected territory, and at the same time a rise of temperature of several degrees is noted in the zone supplied by the nerve" (Sicard).

Immediately after the operation the fingers of the patient are separated from each other by thick layers of cotton-wool, the hand is put in a good position and wrapped up in a thick layer of cotton-wool on which a plaster bandage is applied.

The plaster should be kept on for two to three weeks, then the hand is massaged gently every day, and the fingers are fixed one day in flexion and the next day in extension.

A light and easily movable prosthetic apparatus can be used with advantage.

Daily supervision is essential; healing takes several weeks or even months, and requires a lot of patience.

The paralysis resulting from alcoholisation of the nerve is only transitory, since, according to Sicard's researches, with a solution of alcohol at 20° reaction of degeneration is rare and transient, and regeneration of the nerve takes place rapidly.

CHAPTER XI

PARALYSES AT A DISTANCE. MUSCULAR DYSTROPHIES

Henri Claude, Vigoureux, and Lhermitte in October 1915,[1] under the name of *muscular dystrophy of the myopathic type*, described a form of paralysis with muscular amyotrophy limited to the trapezius and serratus magnus, sometimes on one side of the body and sometimes symmetrical and involving both shoulders.

The remarkable feature in the four cases recorded by these authors is, that they deal with paralysis at a great distance from the wound.

The projectiles (bullet or shell fragment) on their passage through the tissues did not affect the nerve fibres of the paralysed muscles. In one case, even where there was bilateral paralysis of the trapezius and serratus magnus, the original wound was in the lumbar region.

In the different cases related by these writers, amyotrophy, static deformity, and functional disability were marked. One shoulder was lower than the other, and the scapula was rotated so that its axillary border had become horizontal and its spinal border receded from the line of the spinous processes.

Abduction of the arm was very defective, and its forward movement was accompanied by marked winging of the scapula.

[1] Henri Claude, A. Vigouroux, and J. Lhermitte, "Sur certaines dystrophies musculaires du type myopathique consécutives aux traumatismes de guerre," *Presse médicale*, October 11, 1915.

Electrical reactions were only quantitatively modified without any qualitative manifestation of R. D.

Taking all these symptoms into consideration, and especially the localisation of the loss of power in the muscles of the shoulder, particularly in the trapezius and serratus magnus, which are the favourite seat of primary myopathies, and in view of the normal state of sensation and of the reflexes as well as the absence of any reaction of degeneration, the writers reject any hypothesis of a neuritic process, and conclude that this condition must be due to a development under the influence of traumatism of an "impairment of nutrition of certain muscles giving rise to atrophy and secondarily to paresis."

Subsequently Claude, Vigouroux, and Lhermitte [1] reported two other cases of paralysis of the trapezius and serratus magnus, one following a quite superficial wound of the deltoid region of the same side, and the other a fall while the patient was carrying a load. In the last case paralysis developed slowly, and was preceded by pain which was mistaken for rheumatism.

This paralysis assumed features which made it resemble *muscular dystrophy.*

Further, Claude and Lhermitte [2] have had occasion to observe other kinds of paralyses at a distance which they consider to be of a different type, and which they have attributed to lesions of the *peripheral nerves.*

In one of these cases a soldier had been wounded in the internal aspect of the right thigh by fragments of a bomb, portions of which had reached the femur and caused protracted suppuration (nine months). When the patient was able to get up he was paralysed in both legs. The wound, which could only have

[1] Henri Claude, Vigouroux, and J. Lhermitte, "Deux nouveaux cas de dystrophie musculaire à type myopathique consécutifs au traumatisme," *Société Médicale des Hôpitaux*, February 11, 1916.

[2] Henri Claude and Jean Lhermitte, "La névrite motrice extenso-progressive dans les lésions traumatiques des nerfs périphériques," *Société Médicale des Hôpitaux*, July 7, 1916.

involved the muscular and cutaneous branches of the right anterior crural and obturator nerves, had at the end of several months given rise to an atrophic paralysis with sensory and reflex disorders, and a reaction of degeneration in the whole territory of the right great sciatic, and also in the terminal branches (external and internal popliteal) of the great sciatic on the left side.

A second case was that of a soldier who, after a wound of the foot which had completely healed, had shown paralysis of the muscles supplied by the external popliteal nerve of the same side, with reaction of degeneration and *loss of the ankle-jerk*, but without any painful manifestations.

Though we do not pretend to offer a definite opinion as to the nervous or muscular pathogeny of these different affections, we are nevertheless inclined to believe that there exists a close connection between these two types of lesions of which Claude, Vigouroux, and Lhermitte have recorded examples.

Perhaps some obscure affection of the nerves must be incriminated in each of these cases.

The personal cases of which we shall give a short account may serve as a connecting link with the extreme cases mentioned above, and clearly show that we have to deal with one and the same process which as yet is not fully understood.

We have seen three cases of unilateral paralysis of the trapezius and serratus magnus, the first following an attack of rheumatism of the shoulder (according to the diagnosis on the case sheet), the second from carrying a heavy burden fixed by a strap to the shoulder, and the third which had occurred after a wound in the scapular region.

The symptoms were identical with those described by Claude, Vigouroux, and Lhermitte. The following cases are less schematic:—

A soldier who was in perfect health was injured by a revolver bullet, which penetrated the posterior part of the inner wall of the left axilla at a level of

the inferior angle of the scapula ; passing under the superficial layers of the back the bullet came to a stop under the skin at the level of the spinal border of the right scapula. It was easily extracted two months afterwards, and there was hardly any suppuration.

At the time of injury the patient did not fall, had no shock or contusion, and did not spit up any blood. Four or five days after the wound he started to have pain in his arm. On the left side the *pain* ran from the shoulder down to the thumb, and on the right from the shoulder down to the elbow. Fifteen days later he became hoarse and out of breath, and the left pleura was tapped several times, about 2 litres of a blood-stained fluid being removed.

It was only about four weeks after being wounded that his left arm began to become paralysed.

When we examined him two months after the first of the symptoms we found considerable atrophy of the left arm, with complete musculo-spiral paralysis, paresis of the biceps, and flexor longus pollicis and atrophy of the first interosseous space.

Clinical examination showed paralysis of the right serratus magnus and trapezius, with winging of the scapula and paralysis with atrophy of the triceps and supinator longus.

Electro-diagnosis revealed partial reaction of degeneration in all the muscles supplied by the left musculo-spiral and in the first dorsal interosseous.

On the right side there was hypo-excitability to the faradic current and sluggishness of the contraction to the galvanic current in the supra- and infraspinatus muscle, rhomboideus, and lower bundles of the trapezius. The other paralysed muscles only showed quantitative modification in their electrical excitability.

The *reflexes* were affected as follows :—On the right percussion of the triceps tendon caused slight *flexion* of the forearm on the arm, and a brisk contraction of the deltoid. The stylo-radial reflex pro-

voked a contraction limited to the biceps, with slight supination of the forearm. All these reflexes were abolished on the left side.

There was no more pain, but on palpation the musculo-spiral nerve was found to be increased in size and rolling under the finger.

The pain at the first was situated in the course of this nerve.

The disturbance of objective sensibility was very slight, and occupied the territory of the musculo-spiral and musculo-cutaneous.

There was no sign of a spinal lesion.

One year after injury the paralysis, electrical disturbances, and amyotrophy had nearly all disappeared. The patient's voice had again become normal.

Here is another case recalling what A. Hesnard has described as symmetrical irradiation.[1]

P—— was wounded on December 4, 1914, by a projectile in the right deltoid region. Fracture of the humerus. Immediate paralysis of the right musculo-spiral.

Three or four days after the injury there was very severe *pain* in the right biceps, and a short time afterwards a left musculo-spiral paralysis appeared.

Examination fourteen months after the injury (for the report required before payment of the pension) the patient showed a fracture of the right humerus, which was united but still *suppurating*, and a musculo-spiral paralysis from which he had in great part recovered, both from the point of view of voluntary motility and electrical contractility.

There is still, however, some stiffness of all the joints of the limb, and objective sensory disturbances in the first three fingers of the right hand. These disturbances are due to the existence of an arterio-venous aneurism, which clinical examination shows to be in the upper and internal region of the arm. On the left side he shows musculo-spiral paralysis,

[1] A. Hesnard, "Les irradiations symétriques dans les lésions traumatiques des plexus nerveux," *Presse médicale*, May 18, 1916.

with marked atrophy and loss of faradic and galvanic excitability of the paralysed muscles.

In a third patient we saw co-existing with *left musculo-spiral paralysis* (as a result of the fracture of the humerus by a projectile, which *suppurated* considerably and took a long time to consolidate) a paralysis with *atrophy of the right deltoid* and *latissimus dorsi*.

The functional impairment resulting from this last paralysis was more inconvenient to the patient than the left musculo-spiral paralysis.

The fourth case was still more curious. It occurred in a man who had been repatriated from Germany, and who had suffered from a severe wound of the upper part of the *left leg*, with long-standing *suppuration* and ankylosis of the left knee, which was followed by paralysis and atrophy of the muscles supplied by the *left ulnar* nerve.

This paralysis had developed gradually without pain six months after the injury, and had gone on increasing. When we saw the patient fifteen months after being wounded he showed considerable atrophy and paralysis of the interossei and hypothenar muscles, with paralysis of the deep flexors of the last two fingers and the flexor carpi ulnaris.

Reaction of degeneration and well-marked sensory disturbances were present.

In none of these cases had clinical examination shown any involvement of the spinal cord.

We consider that these different paralyses were the result of a neuritic process.

In three cases there had been prolonged suppuration of the original wound.

On two occasions paralysis had been preceded by pain.

Since cases of this kind are still very uncommon, we will confine ourselves to mention them without drawing any conclusion as to their pathogeny.

Treatment must consist as in wounds of the nerve-trunks, in massage and electrization. With regard to

the *measures to be adopted* from the administrative standpoint, we think that, according to the date of the wound, these patients should either receive a temporary discharge or a discharge under medical supervision, with an allowance liable to be renewed. One of our patients had, in fact, almost completely recovered one year after the onset of the symptoms, and another had considerably improved within a similar period.

CHAPTER XII

JOINT AFFECTIONS ACCOMPANYING NERVE LESIONS

In this chapter we shall not discuss ankyloses due to fractures within or near the joints, but shall deal with joint affections encountered in the course of nerve injuries, and which are due to causes which are sometimes difficult to determine.

Generally immobilisation of the limb is regarded as the primary cause of stiffness of the joint. This important causal factor in ankylosis must of course be taken into account. But, as a matter of fact, joint affections only follow immobilisation under certain conditions, which vary with the nerve involved.

The main factors in causing articular rigidity appear to be :—

1. Fracture of a bone, even if it is situated at a great distance from the affected joint.
2. Prolonged suppuration of the wound, especially long-continued suppuration of the area of a fracture.
3. Prolonged immobilisation by an appliance or a sling, especially if the fracture has taken long to unite (a frequent cause of stiffness of the shoulder).
4. Contracture of the muscles acting as active ligaments to the various articulations.
5. Contractures of the ischæmic type, resulting in retraction of muscular tendons (vascular lesions).
6. Various nervous lesions.

The Frequency and Seat of Joint Affections vary according to the Nerve Injured

Musculo-spiral paralyses are very frequently accompanied by ankylosis of the elbow. This ankylosis occurs with the joint in flexion. Extension is limited, and there is often limitation of supination, and sometimes even of pronation. At other times it happens that supination alone is limited, other movements of the elbow being well performed.

During the course of musculo-spiral paralyses, rigidity of the *wrist*, with limitation of extension, may also be seen; occasionally, but more rarely, lateral movements of the wrist are impaired.

Finally, *stiffness of the fingers* is also frequently met with in musculo-spiral paralysis. It is impossible to flex the phalanges passively and completely.

Careful examination of the cases with injury to the musculo-spiral nerve shows certain details, which should be remembered.

They are nearly always cases of fracture of the humerus in its middle third, which have necessitated prolonged immobilisation of the arm, and which have *suppurated*. Ankylosis becomes very gradually established.

More rarely ankylosis was immediate, the elbow having assumed a right angle directly after the wound, and in those cases it can be found on careful examination that the biceps is contractured, its tendon is prominent, and its body may even appear more excitable to mechanical and electrical stimulation.

It is not rare to see cases of injury to the musculo-spiral nerve with a fracture of the shoulder, which have suppurated for a long time, or resulted in pseudarthroses, showing a certain degree of stiffness of the shoulder.

Wounds of the median nerve are only rarely accompanied by ankylosis of the elbow. When this ankylosis exists it is slight, and mainly affects the

movements of supination; occasionally, but far less, that of pronation.

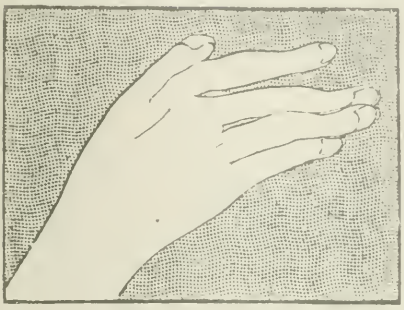


Fig. 54.—Painful form of wound of the median. State of the hand twenty months after the wound. Numerous ankyloses and deformity of the terminal phalanges and of the nails.

Extension of the wrist may be limited; at the same time difficulty in flexing the phalanges on each other is observed.

We have just spoken of complete lesions of the median or of incomplete painless lesions. For in causalgic forms of wounds of this nerve joint affections reach quite unexpected proportions. We have discussed these conditions at length elsewhere.[1] These manifestations affect the elbow joint (ankylosis in flexion), wrist joint (ankylosis in flexion), and especially the finger joints. The articulations most affected are the metacarpo-phalangeal and then the interphalangeal. In the attenuated painful forms these last articulations are the only ones involved (figs. 54 and 55).

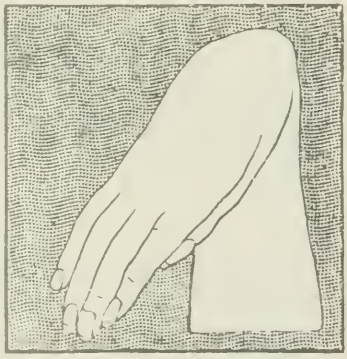


Fig. 55.—An appearance of the hand in the painful form of wounds of the median nerve. Deformity of the fingers and nails. Ankylosis of the wrist in hyperflexion.

In *wounds of the ulnar* we must distinguish between complete painless paralyses and incomplete paralyses which are more or less painful. In the former, apart from the retractions found in ulnar claw-hand, joint affections are infrequent, except where the wound has suppurated for a long

[1] *Clinical Forms of Nerve Lesions.*

period, or where the lower end of the humerus has been involved, with injury to the internal condyle.

On the contrary, in incomplete lesions of the nerve giving rise to painful symptoms it is not rare to see multiple articular rigidities arise in spite of early mobilisation of the limb.

These conditions affect the elbow and the wrist, but principally the metacarpo-phalangeal and the inter-phalangeal joints.

Fig. 56.—Claw-toe deformity following a lesion of the trunk of great sciatic. (Painful form.)

In the course of severe lesions of the *great sciatic* and of its external branch of bifurcation the *external popliteal*, ankylosis of the knee or ankle is hardly ever seen.

It is only the phalangeal joints of the toes which may be affected by a certain degree of stiffness.

But very marked joint affections are caused by incomplete lesions of the trunk of the sciatic, especially those which involve and irritate the fibres of the internal popliteal.

Ankylosis of the knee is very frequent, and is occasionally very marked, but what is seen still more frequently is ankylosis in extension of the tibio-tarsal and metatarso-phalangeal joints, those of the big toe being the most affected.

The articulations of the tarsal bones with each other and with the metatarsals may also become involved, which explains the rolling up of the foot towards its internal border, and the rotation which the front of the foot seems to undergo on the back of the foot (fig. 56).

From this brief summary it appears that incomplete lesions of the median and sciatic nerves, the very lesions which give rise to the causalgic syndrome,

have the most pernicious effect on the joints, and especially on those of the extremities.

Besides which, in fairly numerous cases, the joints of the fingers become thickened, and their swelling contrasts with the thinning of the inter-articular digital segments. Sometimes radiography shows not only decalcification of the bones of the hand or foot, but in addition a very slight increase in size of the bony extremities of the phalanges, and especially of the heads of the metacarpal bones.

It is therefore obvious that the nature of the nervous lesion constitutes a highly important factor.

In many cases of this kind it is the only cause, and neither a fracture of the limb nor a prolonged suppuration can be incriminated. Muscular contracture, ischæmic retraction, and vascular lesions may also be diagnosed.

Ankyloses of the elbow are chiefly met with in lesions of the musculo-spiral, even when the projectile has not fractured the humerus, and the arm has not been immobilised for a considerable period.

It must be remembered that the most frequent site of wounds involving the musculo-spiral nerve is the middle third of the arm, and at this level the nerve has already given off fibres for the triceps, and the majority of its articular fibres for the supply of the elbow.

The bony and muscular lesions, and the formation of more or less developed sclerotic tissue, inflame and irritate the healthy nerves in this situation, hence the articulation which they supply is affected also.

If, as seems likely, these incomplete, painful, and irritative lesions of the nerve are of an inflammatory nature, Guillain's hypothesis, according to which microbial and cellular poisons follow the nerve fibres, and especially their lymphatics, would also, in our opinion, explain the frequent involvement of the articulations.

The synovial sheath of the articulations supplied by

the injured nerve then becomes the seat of a subacute inflammation. This inflammation is propagated to the bony surfaces with trophic impairment and implication of the whole fibrous peri-articular capsule. We know little as yet about what may be the effect of the lesion of the nerve on ligaments and aponeuroses, which depend upon it for their nerve-supply.

With regard to aponeuroses, Henry Meige [1] has drawn attention to an affection peculiar to the knee in patients wounded in the anterior crural nerve, consisting in a loss of power due to a kind of relaxation of the various aponeuroses of the knee, especially those in connection with the vasti muscles and the ligaments of the knee joint. This relaxation persists long after the nerve and the muscle have in great measure recovered.

Prolonged suppuration and immobilisation in the case of a fracture (*e.g.* a fracture of the humerus) affect the distant articulations of the wrist and fingers, the resistance of which have been lowered by the immobilisation of the hand, which the patient cannot move on account of the inconvenience and pain caused by the fractured arm.

These articular disturbances have a bad effect on the nutrition of all the neighbouring tissues; not only do the bones become decalcified, but all the tissues become emaciated, so that the hand assumes an aspect similar to that seen in causalgic hands.

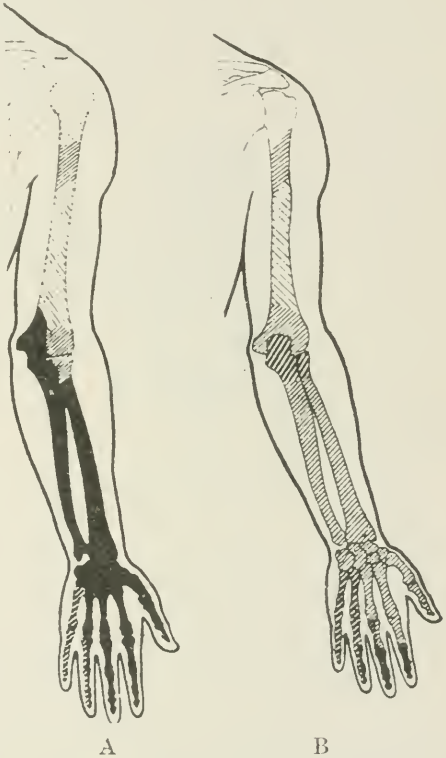
Deformities of the joints due to nervous causes, and especially to irritative traumatic lesions of the nerves, were described some time ago by Weir Mitchell. Professor Dejerine has recently reported two cases of this kind, which are of considerable interest.[2] One of the articular lesions resulted from an irritative lesion of the median "exactly limited

[1] Henry Meige, "De certaines boiteries observées chez les blessés nerveux," *Revue Neurologique*, November–December 1915, p. 939.

[2] J. Dejerine and E. Schwartz, "Déformations articulaires analogues à celles du rhumatisme chronique, avec troubles trophiques, cutanées et hypéridrose relevant d'une lésion irritative du nerf médian," *Société de Neurologie*, meeting of February 4, 1915. M. and Mme. Dejerine and

to the area of this nerve." In the other case the joint lesions, which resembled those of chronic rheumatism, were bilateral, and appeared to be the result of an elongation of the roots of the brachial plexus. This condition arose in the case of a patient suffering from quadriplegia following a vertebral lesion of the lower cervical and upper dorsal region caused by a bullet.

We consider it advisable to end this chapter by a short account of the nerve-supply of the bones and joints of the upper and lower limbs, as a knowledge of this subject may often explain the pathogeny of certain ankyloses.



FIGS. 57 and 58.—Nerve-supply of the bones and joints of the upper limb.

A, anterior aspect. B, posterior aspect.

The circumflex nerve furnishes two articular branches, one of which is given off not far from its origin, and supplies the anterior aspect of the capsule of the shoulder joint.

The second is intended for the lower and internal part of the articulation, and is given off at the part where the circumflex winds round the neck of the

M. Mouzon, "Troubles trophiques articulaires analogues à ceux du rhumatisme subaigu et semblant consécutif à un tiraillement des racines des plexus brachiaux chez un soldat atteint de paraplégie traumatique," *Société de Neurologie*, meeting of July 1, 1915.

humerus; finally, *deltoid branches* furnish osseous fibres for the head of the humerus.

The musculo-cutaneous nerve gives off:—

(*a*) The *nerve to the diaphysis of the humerus*, a motor nerve containing also a few sensory fibres [1] for the periosteum and for the bony substance of the humerus. It accompanies the vessels of the diaphysis and enters the nutrient foramen of the bone. The filaments are lost in the compact tissue and in the marrow of the bone.

(*b*) *The anterior articular nerve of the elbow*, which arises separately or as one of the branches of the nerves to the biceps (Cruveilhier), passes down in the neighbourhood of the sheath of the vessels of the arm, and is distributed to the anterior ligaments of the elbow joint.

(*c*) *A small vascular branch* for the wrist joint. (It arises from the anterior and internal branch of the nerve.)

The median nerve supplies:—

(*a*) A small *articular fibre for the elbow*, which is given off about the middle third of the arm and travels down in the sheath of the brachial artery, which it accompanies up to the epitrochlea. There it subdivides into two filaments which supply the antero-internal part of the capsule of the elbow.

(*b*) *Filaments from the upper branch to the pronator teres*, which are very slender, are also closely applied to the brachial artery, and are distributed to the anterior surface of the elbow joint, or, more precisely, to the antero-external part of the capsule, where they anastomose with filaments of the musculo-spiral.

(*c*) From the *anterior interosseous nerve* arise:—

(1) A small filament for the articular ligaments of the elbow near the head of the radius.

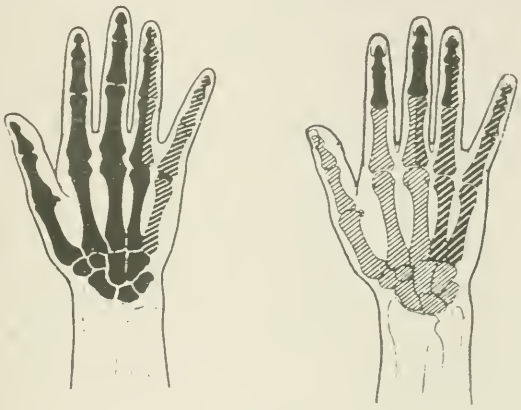
(2) Filaments for the interosseous membrane and the periosteum of the anterior surface of the two bones of the forearm (on the course of these nerves numerous Pacinian corpuscles are found).

[1] Poirier and Charpy, *Traité d'anatomie humaine*, tome iii, fascicule 3.

(3) Branches for the articulation of the *wrist* and for *the first row of carpal bones.*

According to Rauber, the anterior interosseous nerve also gives off nerves to the diaphysis of the two bones of the forearm.

(*d*) *The palmar collateral nerves of the median and their dorsal branches* supply bony and articular filaments. Throughout their course these collateral



A B

FIGS. 59 and 60.—Nerve-supply of the bones and joints of the hand.
A, palmar aspect. B, dorsal aspect.

nerves show groups of Pacinian corpuscles near the articulations.

The ulnar nerve furnishes :—

(*a*) An *articular filament* for the postero-internal part of the elbow joint. It is given off about the middle of the arm.

(*b*) Two *articular branches*, which are given off between the olecranon and internal condyle, and spread out on the articular capsule near the olecranon.

(*c*) Small branches for the dorsal ligaments of the articulations of the carpus. They are given off from the *dorsal cutaneous branch* of the hand.

(*d*) *Digital nerves* and *palmar collateral nerves* given off by the ulnar supply, like those of the median, fine

fibres for the neighbouring articulations. Several Pacinian corpuscules are attached to these fibres.

(*e*) The *deep branch of the ulnar* gives off in its turn branches for the anterior ligaments of the carpus and for the metacarpo-phalangeal joints.

The musculo-spiral nerve gives off :—

(*a*) A recurrent branch for the shoulder joint coming from the branch to the *long head of the triceps.*

(*b*) Filaments given off by the branch to the inner head of the triceps and ending in the internal region of the elbow joint.

(*c*) *Periosteal filaments* for the shaft of the humerus.

(*d*) *Articular filaments* arising at the level of the elbow and distributed to the antero-external ligaments of the elbow joint.

(*e*) The *posterior interosseous nerve*, a branch of the musculo-spiral, gives off filaments to the interosseous membrane and the posterior aspect of the periosteum of the two bones of the forearm, besides which it supplies all the articular nerves of the dorsum of the hand and the root of the fingers.

These nerves also supply the metacarpo-phalangeal joints, after having joined up with the perforating branches of the deep branch of the ulnar.

The nerve-supply of the joints of the upper limb is summarised in the appended table : [1]—

1. *Shoulder Joint.*

Posterior region—Suprascapular nerve.
Anterior ,, —Circumflex nerve.

2. *Elbow Joint.*

Antero-external region	Upper part—Musculo-cutaneous.
	Middle part—Musculo-spiral.
	Lower ,,
Antero-internal region	Upper part—Median.
	Lower ,, ,,

[1] A. Soulié, *Les nerfs.* In Poirier, tome iii. fascicule 3, p. 946.

Postero-external region { Upper part—Musculo-spiral. / Lower part—Ulnar.
Postero-internal region { Upper part—Ulnar. / Lower .. ..

3. *Wrist Joint.*

Anterior region—Median.
Posterior ,, —Musculo-spiral.

4. *Carpal Joints.*

Anterior region—Ulnar.
Posterior ,, —Musculo-spiral.

5. *Finger Joints.*

A. Metacarpo-phalangeal joints.

Anterior region { Filaments of the palmar collateral of the fingers. / Branches of the deep branch of the ulnar.
Posterior region { Dorsal collateral filaments of the fingers. / Dorsal interosseous nerves of the radial.

B. Articulations of the first and second phalanges.

Anterior region—Collateral palmar filaments.
Posterior region { Filaments of the dorsal and palmar collateral.

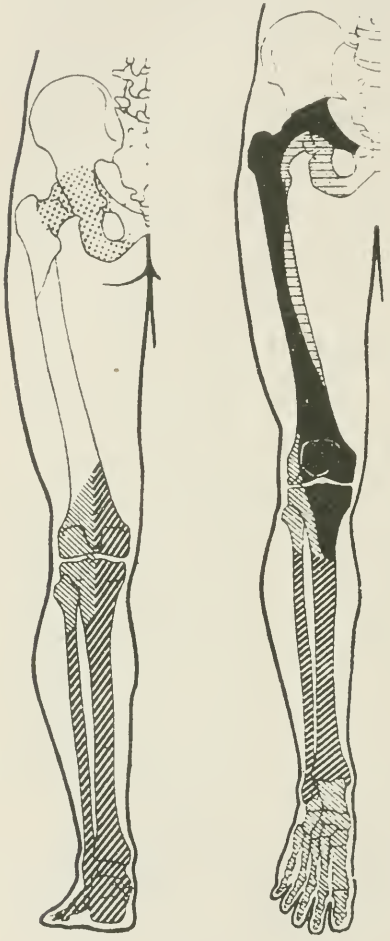
C. Articulations of the second and third phalanges.

Anterior region—Palmar collateral filaments.
Posterior region { Dorsal branches of the palmar collateral, except for the last two fingers.

The nerve-supply to the joints of the lower limb can be summarised as follows :—

1. *The hip joint* is supplied by four nerves—the

anterior crural, the obturator, the sciatic, and the inferior gluteal.



A B

FIGS. 61 and 62.—Nerve-supply of the bones and joints of the lower limb.

A, dorsal aspect. B, ventral aspect.

Great sciatic in white.

The anterior crural nerve is distributed to the anterior and external part of the joint, and especially to the ligament of Bertin.

The obturator gives filaments to the lower and inner part of the joint.

The sciatic and the inferior gluteal supply the posterior region of the hip joint.

2. *The knee joint* receives its nerves from the anterior crural for its anterior surface, and especially from the branch to the vastus externus, externally, and from the internal saphenous on its inner aspect.

The nerves for the posterior part of the capsule are supplied by the internal popliteal for the internal portion, and by the external popliteal for the external portion.

3. *Tibio-tarsal joint.* Three nerves supply this joint — anterior tibial, posterior tibial, and the external saphenous.

The anterior tibial supplies the anterior ligament of the joint, the external saphenous the

external and posterior part of the articulation, and the posterior tibial the postero-internal region.

4. *The articulations of the foot.* Those of the tarsus, like those of the metatarsus, receive their nerve-supply on the dorsal aspect of the foot from a deep branch of the anterior tibial, and on the plantar side from the two plantar nerves, the internal on the inner side, and the external on the outer side.

Explanation of Figures 57 to 60.

In figures 57 to 60 the muscular or cutaneous territory of each nerve is distinguished by special markings, as follows :—

Median and *anterior crural*: solid black.

Musculo-spiral: fine, close cross-hatching from right to left.

Musculo-cutaneous of the arm and *external popliteal*: open cross-hatching from left to right.

Ulnar and *internal popliteal*: thick cross-hatching.

Circumflex and gluteal nerves: black dots.

Obturator: dots separated by lines.

SECTION III

HYSTERIA OR PITHIATISM

AND REFLEX NERVOUS DISORDERS IN THE NEUROLOGY OF WAR

HYSTERIA OR PITHIATISM

PART I

HYSTERIA: PITHIATISM

CHAPTER I

HISTORICAL NOTE

It seems unnecessary for our purpose to give a complete history of hysteria and to repeat what can be found on this subject in most text-books of medicine.

We will limit ourselves to relating the views which were current twenty-five or thirty years ago and those which were adopted later by the great majority of neurologists, especially after the discussions "on the revision of hysteria" which took place in 1908 at the Paris Neurological Society (47).[1]

Has "the modern conception of hysteria" been confirmed by observations made since the beginning of the war? Or should these observations, as some think, lead us back to some extent to the "old conception"?

To enable the reader to form an opinion on this question he must know the essential points of these two conceptions of hysteria. We will describe them briefly, but without omitting any details which are needed to illustrate their different characteristics.

[1] We have given in the bibliography the list of the principal publications dealing with the questions discussed in this book. All are numbered, to correspond with the figures inserted in the text.

The Old Conception of Hysteria

According to this doctrine hysteria is manifested by two varieties of disorders. The first, which are permanent, are known as "stigmata"; while the second, though they may persist for a long time, are usually transient, being liable to be provoked or suppressed by external influences or purely psychical causes.

Some of the manifestations may be associated with or substituted for others and appear in the most varied guises. Neither of these two varieties causes any grave disturbances in the general nutrition or the mental state.

Some hysterical disorders have a characteristic appearance which enables the observer to recognise them without paying any attention to the accompanying symptoms or the circumstances in which they arise. The following are examples: *Hysteria major* with its four periods, epileptoid period, period of contortions and generalised movements, period of attitude expressing various passions, and period of delirium, is absolutely characteristic, whatever views may be held as to its mechanism; no other affection but hysteria can give rise to such disturbances. Much more common than hysteria major is *Hysteria minor*, which is an incomplete form of the preceding, and includes almost countless varieties. Though generally easy to recognise at first sight it may be mistaken for epilepsy, if the character of the movements be taken into consideration alone; sometimes the attacks succeed each other uninterruptedly and constitute a sort of *status hystericus* comparable to the *status epilepticus*. Nevertheless, whatever its form, duration and intensity may have been, the attack has no serious results. In spite of the convulsions and contortions which astonish and alarm the laity, there is hardly any risk of severe wounds or contusions, as in the case of an epileptic fit. It has never been seen to occur in sleep or to cause an involuntary emission of urine, subconjunctival ecchymoses or subcutaneous hæmorrhages. It is not

followed by a long period of prostration or torpor; on recovery from the attack the patient has only a slight feeling of discomfort, and almost at once recovers his lucidity. Even when prolonged the status hystericus does not produce the rise of temperature which is so common in the status epilepticus.

In *hysterical hemiplegia* the gait, as described by Todd, has a peculiar character which enables it to be recognised at first sight.

"The patient," says this eminent English clinician, "drags the palsied limb after her as if it were a piece of inanimate matter, and uses no act of circumduction nor effort of any kind to raise it from the ground: the foot sweeps the ground as she walks. This, I believe," he adds, "is characteristic of the hysterical form of paralysis."

Hysterical hemiplegia like all other hysterical paralyses may remain without any change in its appearance for months and even years—an occurrence unknown in organic paralysis, which never remains the same from the beginning to the end, but follows a definite course.

Hystero-traumatic brachial monoplegia, which was first observed by Charcot in a man, and described by him in his celebrated lectures, is identical with that which he was able to reproduce by hypnotic suggestion. One of its attributes is the association of an anæsthesia of the upper limb "resembling a shoulder of mutton in its distribution," with slight involvement of the thorax in front and behind.

In *glosso-labial hemispasm*, described by Charcot, Brissaud and P. Marie, the spasm, as its name indicates, may be limited to the tongue and lips but sometimes affects simultaneously the orbicularis palpebrarum, platysma and neck muscles. The hook-like appearance of the tongue and the intermittent spasms of the contracted muscles give it an almost pathognomonic appearance.

Astasia-abasia, which has been specially studied by Charcot, Richer and Blocq, who gave its name to the

condition, consists in a peculiar dissociation of the motor functions of the lower limbs. In a lying or sitting position the patient can move his limbs freely without a sign of anything being wrong. He is even able to walk on all fours, to jump and climb. But he is incapable of standing or walking normally; sometimes these two functions are abolished or weakened (paralytic or paretic forms), sometimes they are disturbed by abnormal movements (ataxic, choreiform or tremulous forms).

This dissociation of the motor functions of the lower limbs is peculiar to hysteria. When showing a woman suffering from *hysterical mutism* at one of his Tuesday lectures Charcot expressed himself as follows—

"Not only has it become impossible for her to articulate a single word in a loud or a low voice, but she is even incapable, in spite of all her efforts, of emitting any laryngeal sound or noise whatever. It is true that she will still have a means of communicating with us by an intelligent pantomime or what is still better by writing, for although there is an absolute motor aphasia, the faculty of expressing herself by writing has, in striking contrast, remained perfectly unaffected, as you will see in a moment. Gentlemen, even from the rapid description I have just given, those of you who have attended these lectures for the last three years will have at once grasped that we are dealing with a case of hysterical mutism. In no other condition, in fact, will you find this combination of characteristic symptoms."

We will also quote the following passage from Charcot's Tuesday lectures—

"Rhythm and cadence are characteristic of a number of hysterical phenomena, as I have several times had occasion to remark. In *rhythmic chorea* especially this feature is so pronounced that a dancing-master could follow and record the peculiar and often very complicated movements carried out by the patients during these attacks. As Hamlet says,

there is ' method in their madness.' The same applies to hysterical coughing, lowing and barking. I believe that it may be asserted that all yawning recurring at regular intervals as in the present case, is an hysterical phenomenon."

Although hysteria is often manifested by symptoms which it alone is capable of producing, it can also assume the appearance of various other affections and even of organic disease. It is able to produce motor disturbances which in themselves are indistinguishable from those caused by cerebral or spinal changes.

" Convulsive hysteria," said Brissaud (1), " is an affection in which the functional disorders of the pyramidal tracts play an important part, and we could have shown cases of permanent hysterical contracture which entirely resemble those of hemiplegic or paraplegic contracture."

" The tendon reflexes," said Richer, " are usually exaggerated, and the paralysis assumes all the characters of spastic paralysis, the most important of which are exaggeration of the tendon reflexes and ankle clonus. Thus by insensible degrees paralysis may be transformed into contracture, and we have had an opportunity of seeing in hysteria cases of spastic paraplegia which gradually assumed all the characters of permanent contracture of the lower extremities with the closest resemblance to ataxic paraplegia due to an organic lesion. Circumstances of this kind may cause considerable difficulty in diagnosis."

Hysteria has been considered capable of producing superficial or deep lesions and disturbances of all kinds : such as erythemata, vesicles, bullæ, hæmorrhages, ulcerations, and even gangrene of the skin, and subcutaneous œdema, laryngeal paralyses, spasm of the glottis, œdema of the glottis, pulmonary congestion and hæmoptysis ; gastric ulcer, hæmatemesis and melæna ; anuria, hæmaturia, albuminuria ; angina pectoris, tachycardia or bradycardia ; and fever which sometimes may be very high.

Hysteria would thus occupy a very extensive domain, the limits of which could not be fixed. "Hysteria," as Sydenham said long ago, "imitates almost every disease which befalls mankind. Whatever part of the body it attacks, it will create the proper symptom of that part. Hence without skill and sagacity the physician will be deceived, so as to refer the symptoms to some essential disease of the part in question and not to the effects of hysteria."

Hysteria is a kind of Proteus, and has been called "the champion imitator."

How is it possible to find the true cause of nervous symptoms when their appearance is the same whether their origin is organic or hysterical?

It used to be said that the answer to this question was to be found by looking for the *stigmata*. Although, according to Charcot and Pitres, these stigmata are not constantly present, they constitute clear evidence of hysteria. In addition to being permanent they develop without the patient's knowledge. They are as follows: Anæsthesia of the pharynx, hemianæsthesia, consisting, in a well-developed form, of abolition of the various kinds of common sensation and unilateral diminution of the acuity of special senses, especially vision, with concentric narrowing of the visual field, diplopia or monocular polyopia and a dyschromatopsia in which, unlike that seen in tabes and alcoholism, the blue and violet field is affected while the perception of red remains normal.

The various painful areas such as the clavus hystericus in the head may also be reckoned among the stigmata. As regards the etiology, *neuropathic heredity* constitutes the essential and constant factor in hysteria, but its manifestations may be due to a variety of *provocative agents*, such as various intoxications and infections, trauma and psychical influence. Imagination, suggestion (Charcot, Bernheim, Janet, Strümpell) and "mental representations" (Moebius) play an important part in the genesis of hysterical symptoms, which in Russell Reynolds' words are

"dependent on idea." The following, according to Charcot, is the ordinary mechanism of hystero-traumatism. An individual with a predisposition to hysteria receives, it may be, a blow on the shoulder which is insufficient to cause an appreciable lesion. However, although he continues to make use of his arm, the idea of motor impotence occurs to him; it gradually increases during a period of varying duration (phase of meditation), and finally manifests itself in the form of brachial monoplegia.

But, although suggestion in the form of auto- or hetero-suggestion is of considerable importance, the principal factor is usually emotion; it prepares the ground and is even supposed to have the power to create hysterical disorders without consciousness being aware of their development or the will interfering to oppose them.

Permanent stigmata especially were supposed in some cases at least to be independent of suggestion, and some hysterical symptoms were regarded as explicable by a physiological mechanism.

Such was the general conception of hysteria a quarter of a century ago, though this does not mean, of course, that agreement was complete on all points. We need only allude to the discussions which took place between the Salpêtrière School and the Nancy School, though these were principally connected with hypnotism. As regards hysteria, properly so called, the pathogeny of hysteria major with its four periods was specially the object of controversy; some regarded it as a spontaneous expression of the neurosis, occurring without the intervention of any suggestion; while others, on the contrary, such as Bernheim, considered that it represented an artificial creation of which the physician himself was the chief author. Pitres was guarded as to the psychical mechanism which Charcot invoked to explain the genesis of traumatic brachial monoplegia.

"The hypothesis," he wrote, "is an attractive one. It explains in a fairly satisfactory manner the late

appearance of most of the hystero-traumatic paralyses, but it is difficult to apply to every case." Limits of space prevent us from discussing this subject in greater detail.

Modern Conception of Hysteria: Pithiatism

Complete agreement on all questions relating to hysteria has not even yet been reached. But on certain points of primary importance the old views have been abandoned by all neurologists, and the new ideas have been generally adopted, as may be seen from the discussions which took place at the Paris Neurological Society in 1908.

It is therefore justifiable to say that there is a "modern conception of hysteria." But before describing it, we must warn the reader of certain errors of interpretation which have been too frequently committed. Those who have adopted the new conception have been credited by a number of badly informed medical men with the opinion that nothing remains of the old hysteria, that hysteria is indistinguishable from simulation, and has no longer any real existence, so that the dismemberment of hysteria has been a destruction of this protean affection. This is a gross misrepresentation. The most enthusiastic adherents of the modern school are the first to recognise the value of a part of the work of their predecessors. The clinical picture of the various syndromes which we have dealt with in the previous section, such as certain forms of hemiplegia, astasia-abasia, mutism, and rhythmic chorea, has been drawn, as no one will deny, in a masterly manner, and in this respect there is nothing to suppress. We gladly admit that the experimental investigations of Charcot on hystero-traumatic brachial monoplegia have been particularly illuminating, and have helped to establish the psychical nature of hysterical manifestations. After making this correction, we must try to show what has been the development of ideas. As one of us

has been their chief promoter, we may quote from one of his writings on the subject (61) certain passages which will enable the reader to understand how he was guided to form this new conception of hysteria.

"I had at first concentrated my attention on the comparative study of hysterical and organic paralyses, and, as I have already had occasion to remark, I was struck by the inadequacy of the means at our disposal for distinguishing them. I well remember that these two kinds of paralyses were supposed to exhibit the same intrinsic characters; it was only in the concomitant circumstances that the distinctive feature was to be found which they did not possess in themselves. A hemiplegia, for instance, occurring in a young subject who had neither cardiac disease nor syphilis, and accompanied by 'stigmata' of the 'neurosis' (hemianæsthesia, anæsthesia of the pharynx, narrowing of the visual field, etc.), was usually regarded as hysterical, and it was in the reverse circumstances attributed to a lesion of the nerve centres.

"Such reasoning showed a lack of precision and defective logic. It had indeed long been known that in the first place the various manifestations of hysteria were not invariably associated with the so-called stigmata, and secondly that the latter might occur in organic paralysis. The presence or absence of anæsthesia of common or special sensation cannot therefore authorise us to affirm or deny the hysterical nature of a paralysis; just as the presence or absence of signs of a cardio-vascular affection or of syphilis does not warrant us in admitting or rejecting the idea of an organic paralysis. As the result of these considerations I became convinced of the necessity for subjecting paralytic disorders to a more rigorous examination and for endeavouring to find the differential characters inherent in the paralyses themselves.

"Methodical investigations, the first decisive results of which were published in 1893, led me to the con-

clusion that there is no change in the tendon reflexes in purely hysterical paralyses and contractures (22).

"This statement, simple as it appears to-day when it meets with hardly any opposition, was for a long time disputed and periodically, for years, gave rise to hostile criticism. The objections, however, were always due to inaccurate observation or false interpretation of facts, as I was able to show whenever I had an opportunity of testing them.

"On reference to the Proceedings of the Paris Neurological Society cases of this kind are to be found, such as a so-called hysterical hemiplegia accompanied by unilateral exaggeration of the tendon reflexes, or a paraplegia with exaggeration of the reflexes in the lower limbs and ankle clonus also attributed to hysteria. But in none of these cases was the diagnosis of hysteria verified; as a rule those who had shown cases of the kind afterwards admitted that the course of the disease compelled them to abandon their original hypothesis and to recognise, in conformity with my opinion, that it was some organic affection. In other cases a different error was committed: a patient who was really suffering from hysterical paralysis was wrongly supposed to have very exaggerated tendon reflexes and ankle clonus.

"The prevalence of the belief that hysteria like lesions of the pyramidal system might cause exaggeration of the reflexes was principally due to the fact that the study of reflexes in the normal condition had been neglected and that the scale of their individual differences had not been properly determined. It was by establishing an exact scale (179) and by distinguishing 'complete' ankle clonus which is pathological, from 'incomplete' ankle clonus which is physiological, and may of course be observed in an individual with hysterical paralysis as well as in a normal subject, that I supplied the means of avoiding what had so frequently been a stumbling-block to others.

"Hysteria has no appreciable action on the tendon

reflexes; it has no more power to exaggerate them than it has to weaken or abolish them. The same applies to the pupillary reflexes and pharyngeal reflex. These conclusions which result from my investigations and which are to be found in every text-book nowadays, in addition to other new facts, have sapped the foundation of the old hysteria.

"I have described a number of signs which are peculiar to organic paralysis (25) (61).

"I have also been able to show, even since 1892, how an examination of the character of the motor disturbance exclusively is sufficient to distinguish hysterical from organic facial paralysis (18) (20).

"This series of studies, which have been unanimously confirmed, has brought about the first and very important change in the traditional doctrine of hysteria. We may therefore conclude (i) that hysteria is very far from being able 'to do everything' as was formerly stated (33); (ii) that it cannot reproduce certain characteristic phenomena of organic paralysis; (iii) that the diagnosis of these two kinds of paralyses instead of depending on extrinsic characters, and thus being arbitrary and being left to the perspicacity of the doctor, is subject to exact rules, and in most cases can be made with certainty now that it depends on the examination of intrinsic characters.

"But this was not the only consequence of these new discoveries. My conclusions, which I have just stated, were logically bound to lead me still further. I found it necessary to make a general revision of the circulatory, trophic and thermal disorders attributed to hysteria, for the notion that such disorders were caused by hysteria seemed partly to be due to cases of hemiplegia which had been improperly regarded as hysterical. It is not uncommon indeed to find vaso-motor phenomena and hypothermia on the paralysed side in organic hemiplegia; in such a case with an erroneous diagnosis of hysterical hemiplegia one was inevitably bound to conclude that hysteria has the faculty of producing circulatory disorders,

hence to the conclusion that it could give rise to hæmorrhages and other lesions in the skin there was only a step, so why should there not be visceral hæmorrhages also due to hysteria? Hysterical fever likewise appeared to some to be quite a natural occurrence, for if hysteria was able to exercise a disturbing influence on the centres of the tendon reflexes and the vaso-motor centres, there were grounds for supposing that it might also disturb the working of the centres which regulated the temperature. As all these deductions were logical and were apparently corroborated by actual cases, they were accepted by men of the keenest intelligence, but as they originated in error they were absolutely vain and worthless. I therefore thought that a clean sweep should be made, and that an unprejudiced enquiry should be started for new facts relating to this question.

"My personal observations made me doubt more than ever the authenticity of the disorders which I have just described: I did not have an opportunity of seeing a single conclusive example.

"For years I have constantly asked my colleagues to show me cases of the kind wherever they met with them. At first they thought that they had found some, but there was merely a repetition of what had happened in the case of 'hysterical exaggeration of the reflexes': events soon proved that they had made a mistake. Sometimes it was a case of œdema, described as hysterical, which was afterwards shown to be due to tuberculous synovitis; sometimes it was a case of formation of vesicles also attributed to hysteria, which, as was discovered later, were produced voluntarily by the patient by means of artificial irritation. None of these cases could resist a searching examination. Those who persisted in remaining faithful to the old doctrine cherished the hope that they would one day meet with what their predecessors declared they had seen, repeating that 'negative facts cannot prevail against positive facts.'

"For my part, I did not cease to maintain that the

old cases were liable to suspicion and that new ones were needed to settle the problem.

"In order to make my investigations as extensive as possible I recommended one of my pupils, Dr. Mendicini Bono, to address an enquiry on this matter to a large number of medical men on the staff of Paris hospitals. None of them was able to supply a single fact in confirmation of the hypothesis of circulatory or trophic disturbances due to hysteria. Nor, again, was any one found to guarantee their existence when their reality was discussed in 1908 at the Paris Neurological Society.

"There is no doubt that even in days gone by some of the cases described under this title were spurious. Thus, certain forms of 'hysterical fever' were found to be artificial. Charcot himself had issued a warning as to errors of interpretation or fraud. It is none the less true that this eminent neurologist believed in the existence of hysterical hæmorrhages and skin lesions, and that he gave the name of blue œdema to a circulatory disorder which he attributed to hysteria.

"A close examination of the facts has enabled me to establish that there is not a single case justifying such a point of view. Occasionally even now this statement is contradicted, but is not this the case with the most firmly established medical truths?

"To sum up, in former times hysteria was credited with the faculty of producing vesicles, ulceration, superficial gangrene, cutaneous or visceral hæmorrhages and fever; it was also supposed to be able to produce albuminuria and anuria. All this was to be found in the text-books, and there were numerous cases of the kind reported. Since 1901, when my first comprehensive study of the limitations of hysteria appeared, these publications have become increasingly rare, and one would search in vain for a single case in the Proceedings of the Medical Society of the Paris Hospitals or of the Neurological Society for the past

six years. The history of such cases is now nothing more than a legend.[1]

"The result of the work which I have just summarised was to restore what hysteria had unduly appropriated to conditions which to-day are well recognised as organic affections, and also to strip hysteria of the products of simulation. The work in question has a practical value which will be discussed later, and constitutes an acquisition whose importance, I would insist, in no way depends on the opinion that may be held as to the other questions which we will now examine.

"Although the ground had been partly cleared, a large amount of material still remained. This residue was composed of disorders whose common character was the absence of an anatomical substratum, at least in so far as the methods of investigation at our disposal allowed us to ascertain. But this point of resemblance was not enough to justify their complete assimilation.

"Did these different parts form a homogeneous whole to which the term 'hysterical' could be applied? Was it not rather an artificial assemblage composed of heterogeneous elements? Such was the new problem which I set myself.

"I will allude only to those observations and comparative experiments of mine which appeared to me to be conclusive and have allowed me to attain my aim. As I mentioned above, the rôle of suggestion in the production of hysterical disorders, and that of psychotherapy in their treatment, had long been known. Nevertheless, in spite of the importance attributed to these agents, it was generally agreed that their influence did not extend to all the manifestations of hysteria. Was it possible to have any other opinion at a time when organic affections were included in this neurosis?

[1] I exclude for the moment the vaso-motor troubles of a superficial and transitory character (erythema, dermographia), which will be discussed later (*v.* p.422).

"Failure was obviously inevitable when, for instance, a persistent attempt was made to treat hemiplegia due to cerebral hæmorrhage by psychical methods under the impression that it was hysterical. But as the progress of semeiology had supplied the means of avoiding such errors, and the intrusion of organic affections into hysteria was no longer to be feared, I thought it advisable to resume the study of suggestion and counter-suggestion in their relations to the various disorders under discussion. Subsequent investigations enabled me to divide these phenomena into two distinct groups: suggestion produces the first but has no action on the second. To avoid a misunderstanding, however, I must give some complementary explanations.

"The first of these groups includes convulsive attacks, paralyses, various kinds of contractures, tremors, choreic movements, which are sometimes irregular but usually rhythmical, and disturbances of phonation, respiration, common sensations (anæsthesia, hyperæsthesia), and special senses, all possessing the common characteristic of being liable to be reproduced by experimental suggestion which—and this is a most important point—is *capable of determining their form intensity and duration.* These phenomena, it may be said in passing, may be imitated by a clever and well-educated simulator, which constitutes a source of difficulty in medico-legal enquiries relating to what is called 'hystero-traumatism.'

"These disorders are liable to disappear sometimes instantaneously, under the sole influence of persuasion or counter-suggestion. It may even be said that psychotherapy cures them almost infallibly unless it is counterbalanced by an opposing influence, such as the counter-psychotherapeutic action exercised involuntarily by the patient's entourage or in cases of hystero-organic association, by auto-suggestion which is maintained by the disorder due to the lesion. I will add parenthetically that these manifestations may resist treatment in a subject suffering from a

mental affection such as hebephrenia. The second group comprises on the one hand dermographism, which indicates an exaggeration of the cutaneous vaso-motor reflexes, and on the other abnormally intense and prolonged emotional reactions such as tachycardia, erythemata, and over-activity of the sweat or intestinal glands.

" Suggestion has no power to reproduce them and counter-suggestion has no action upon them. I have been met with the retort that it is possible to provoke in some persons, almost at the word of command, an acceleration of the heart-beats and vaso-motor reactions which might be regarded as the effects of suggestion.

" The influence of suggestion, however, in this case is more apparent than real. In reality such an influence is very indirect on disorders of this kind; when suggestion does give rise to them, it is only by the agency of the emotion which it may produce. Suggestion cannot control them : as soon as the disorders appear they escape its influence; it is unable to determine *their form intensity and duration.*

" Thus a fundamental characteristic separates these two groups of disorders. That is a new point which is definitely established.

" However, in spite of their differences it might be possible for them to be connected by some bond of union, and it would be right to maintain this if there was a sort of attraction between them; but as a matter of fact there is nothing of the kind. As I have shown by a comparative study, dermographism and excessive emotionalism are as frequently seen in individuals who are otherwise normal as in those who present symptoms characteristic of the first group, such as fits, contractures, etc.

" There is, therefore, no reason to unite these two groups of phenomena and to give them a common title. They should be completely dissociated and each receive a different name. This conclusion which I have reached is by no means arbitrary, but is logically inevitable.

"It would obviously be unreasonable to continue to give the name hysterical to the disorders ranged in the second group; besides, are not the terms 'dermographia,' 'vaso-motor disorders,' or 'disorders of the vaso-motor reflexes' and 'emotional disorders' sufficient to express the ideas attached to them?

"There might even be some advantage in abandoning the use of the term hysteria, which in its etymological sense is in no way suitable for any of the phenomena under consideration. If, however, it is to be retained, it should be reserved for the first group, comprising those impressive disorders which this word calls to our memory almost automatically (fits, epidemic chorea of the middle ages, and paralysis miraculously cured). I have proposed the substitution of the term 'pithiatism,' from *πείθω*, 'I persuade,' and *ἰατός*, 'curable,' which expresses one of the fundamental characteristics of these symptoms, viz. the possibility of being cured by the influence of persuasion.

"The dismemberment of the traditional hysteria is therefore an accomplished fact: the heterogeneous elements of which it was comprised as the result of an artificial grouping have been separated and each has been put in its natural place. 'Hysteria,' said Lasègue, 'has never been defined and never will be.' Indeed, as it is impossible to give a definition which is applicable to a number of different objects it was impossible in former times to define the conditions comprised under this title. Nowadays, it is different. The isolation of the group of phenomena which may be called indifferently hysterical or pithiatic is an accomplished fact. They possess characteristics which belong only to themselves, which are absent in all other morbid states, and which therefore constitute the elements of the definition of hysteria (26) which I have set forth as follows: '*Hysteria is a pathological state manifested by disorders which it is possible to reproduce exactly by suggestion in certain subjects and can be made to disappear by the influence of persuasion* (*counter-suggestion*) *alone*' (59). Some authors have

criticised this definition, on the ground that persuasion or counter-suggestion is capable of curing neuropathic disorders which are not hysterical, especially neurasthenic symptoms. Such a criticism is obviously due to a confusion between hysteria and neurasthenia. It is agreed that the phenomena of exhaustion which constitute the essential characters of neurasthenia cannot be cured by counter-suggestion alone : the symptoms which are caused to disappear by this method are merely hysterical or pithiatic symptoms which have been grafted on to neurasthenia. In 1892, or twenty-one years ago, I insisted on the frequency of associations of this kind.

"I think then that I can say that this definition is adequate ; it suits the whole of the object defined, and is applicable to it alone.

"The fact that suggestion has the power of giving an exact reproduction of every hysterical disorder undoubtedly does not prove a priori that such disorders are actually due to suggestion. However, as I have previously said, the possibility of its participation in the production of apparently spontaneous hysterical phenomena has never been denied ; clinical observation has so clearly proved this that such a truth could not have escaped notice. But to what extent does suggestion intervene ? In former times one was very far from realising its various modes of action. There was one in particular which for a long time was overlooked, viz. medical suggestion, to which Bernheim attracted attention and of which I have shown the great importance.

"Its influence can be seen in the study of the so-called hysterical stigmata which were said to develop without the patient being aware of them, like an organic disease, and without any previous mental representation. Bernheim himself admitted the possibility of an hysterical hemianæsthesia and amblyopia which were not caused by suggestion. Until recently most hysterical patients showed the so-called 'permanent' stigmata which, according to the old

belief, gave the transitory symptoms a genuine character. This was an error and was due to the fact that the unconscious influence exerted by the doctor's questions and attitude on the genesis of these disorders was not taken into account. I convinced myself of this by the systematic study of a series of patients with hysterical symptoms who had hitherto not undergone any medical examination: I investigated the conditions of their sensation and vision, always avoiding any untimely reflections or gestures during my enquiry which might interfere with the spontaneity of their replies. Now, in none of these patients, who numbered more than a hundred, did I find any hemianæsthesia, narrowing of the visual field, or dyschromatopsia. Comment is needless.

"Suggestion as manifested by a love for imitation offers an explanation of the contagious character of hysterical phenomena, especially nervous attacks, which formerly, when their mechanism was not understood, used to spread like an epidemic through the wards. Suggestion may intervene in a different form in hystero-organic associations: auto-suggestion replacing or accompanying hetero-suggestion. It is easy to understand that in a disease with any tendency to persist the symptoms on which the patient concentrates his attention and apprehension will cause disorders to develop which belong to the domain of suggestion. Sometimes this organic factor may be minimal, but a transient disturbance, a slight pain, a trivial injury, or a mere bruise, in a susceptible individual may be the starting-point of a complex process of auto-suggestion in which psychological analysis might have much difficulty in unravelling the part played by various factors, such as the patient's own reflections, his previous experience and beliefs, the misguided solicitude of his friends, and, lastly, medical examination with its impressive paraphernalia, admirably suited for rousing the patient's attention and directing his thoughts into unexpected channels. All these factors become inextricably mixed, and their

combined action gives rise to the hysterical phenomenon which henceforth persists by itself, whereas the preliminary process of auto- and hetero-suggestion is very likely to pass unnoticed.

"There is no need to insist further on the importance of suggestion in the genesis of hysterical phenomena.

"But I have not yet proved that it is a *sine qua non*. Have not other agents, especially emotion, the power to produce them, as was unanimously believed until quite recently?

"Before attacking this problem it is important that it should be stated in precise terms. No doubt there are grounds for supposing *a priori* that physical commotion and, still more, moral shocks may diminish the personality, weaken the critical sense, and increase the suggestibility, and so play an indirect part in the development of pithiatic disorders; but these agents in such cases would be merely preparing the ground for suggestion. Are they capable by themselves of producing hysterical phenomena apart from any suggestion, as has been maintained? In other words, can an hysterical paraplegia or monoplegia, for example, appear automatically under the influence of an emotion [1] without any preliminary mental repre-

[1] In a work written in collaboration with J. Dagnan-Bouveret, entitled *Emotion and Hysteria*, we insisted on the distinction that should be made between emotion in the sense of a shock and a gradual emotion. We reserved the name emotion for the former, and the latter we called an affective state.

We defined emotion as follows—

"A violent modification of affectivity occurring under the influence of a sudden representation which upsets the physiological or psychical equilibrium during a period which is usually of short duration." We have shown that in the affective life diffuse phenomena must be distinguished from those which are systematised. The first group comprises all the emotions properly so called and certain affective states like vague and unreasonable melancholy. The second group contains the organised affective states whose principal types are sentiments of hope and fear, these states possessing the power of entering into representative complexes and giving them life.

sentation, like a secretion of sweat, an intestinal flux or an erythema? Such is the all-important problem which requires to be investigated afresh by one who wishes to penetrate the nature and mechanism of hysteria, for the method hitherto employed to solve it is defective.

"The method is as follows—

"Given a patient with hysterical symptoms, an attempt is made to determine the circumstances in which they developed by examination of the patient and his friends; if there is a history of an emotion, it is regarded as being the cause of the symptoms in question. Observations of this kind have led to the classical opinion as to the rôle of emotion in hysteria.

"I have shown that by following such a path it is impossible to attain the truth; in a matter of this kind one must view with suspicion the information given by patients who are inclined, even when suffering from an organic affection, to attribute their disorder to an old or recent emotion; with the best faith in the world they may lead one into error. Besides, in the problem before us it was not enough to establish that the development of one of these accidents had been preceded by a psychical shock; it was also necessary to prove that suggestion had nothing to do with its development. Now suggestion implies the idea of a perturbation of consciousness, and any one submitting to its influence cannot have a clear idea of it.

"The method hitherto adopted by which the examination was made by means of *retrospection* cannot lead to the end in view. I have adopted another method which proceeds in an entirely opposite manner; given the presence or absence of certain conditions which are or seem to be suitable for the development of nervous symptoms, an examination is made for any hysterical disorders. This may be called an examination by *prospection*. The patients are not asked to furnish a history or supply explanations of their case. By means of this method, if a

number of observations are made, it is possible to detect the supposed causes of hysteria, to dissociate them and to judge of the value of each. I have already used it in the criticism of 'stigmata,' comparing, as we have seen, two groups of hysterical subjects, one of whom had been subjected to and the other removed from the influence of medical suggestion. The comparison showed that without its intervention these stigmata did not develop: suggestion, therefore, appears to be an indispensable condition for their production.

"This method is also applicable in a comparative study of hospital conditions in the past and present; formerly it was by no means exceptional to see several patients at once in a ward in convulsions with attacks of opisthotonus, a sight never seen nowadays. And yet the human heart is no more secure against moral shocks to-day than it was in former times. Why, then, has this change taken place? The reason is that formerly the doctor unconsciously 'cultivated' hysteria, or at least, as he did not understand its mechanism, he did not take, as he does at present, the proper measures for causing the manifestations to disappear as soon as they develop and to preserve the patient's neighbours from infection.

"Are we not justified in concluding that emotion, or at least emotion by itself, cannot produce hysterical attacks? Similar remarks might be made with regard to epidemics of hysterical rhythmic chorea which have not been seen for a long time.

"The method of prospection finds a direct application, and gives more conclusive results as regards the point at issue in questioning those who by chance or by reason of their profession have been in contact with a large number of persons under circumstances in which emotion was inevitable, and have had an opportunity of observing them at the moment of the shock before the phase of 'meditation,' during which suggestion so frequently comes into play.

"The following is a brief summary of the results of

various enquiries of the kind, the first of which relate to catastrophes in which a large number of persons were simultaneously affected. Several medical men who have been present at serious railway accidents and given immediate attention to the wounded have informed me of their observations, which all agreed with one another; none of them had observed any hysterical disorders.

"Neri's account of the earthquake of Messina is also very instructive. Although he examined more than 2000 persons immediately after the catastrophe he did not see a single case of paralysis, contracture or convulsive attacks. The studies which he carried out later, in the weeks following the disaster, at a time when suggestion might have entered into play and accentuated the phenomena caused by the psychical shock, gave the same results. It is to be noted especially that Neri carefully examined the state of sensation and measured with the perimeter the visual field of more than 600 survivors, some of whom were suffering from various nervous disorders constituting a real morbid condition or 'emotional neurosis'; he did not find a single case of hemianæsthesia or narrowing of the visual field. One is justified, however, in supposing that under the circumstances emotion must have reached its utmost intensity and would be expressed outwardly in a startling degree.

"Personally, I have made enquiries of mortuary attendants in the various Paris hospitals. There can, indeed, be no doubt that when persons see the dead bodies of their relations they are likely to feel a deep emotion, which is increased by the environment in which the scene takes place. Among these enquiries, all of which led to the same result, I will specially mention that which I made in company with my colleague Richardière at the Hospital for Sick Children. During a period of eighteen years, in which more than 20,000 deaths had been entered in the register, the mortuary attendant had seen about 10,000 women come to identify their child's body and be present

when it was put in a coffin; there can be no doubt that he witnessed sincere emotions. Now this man, in his very full replies to our questions, declared that he had never observed a single manifestation of undoubtedly hysterical character; he remembers that only five or six times in his long experience women had a sort of faint and remained unconscious for a few moments, but he had never seen a convulsive attack, and he declares that he had never witnessed a single case of paralysis or contracture. Enquiries of the same kind were made of medical men, sisters and nurses who had several times had occasion to observe the effects of the most violent moral shocks, and all had the same result.

"Investigations by prospection thus present us with an entirely new aspect of the relation of hysteria to emotion: they show that emotion alone, however intense it may be, does not produce hysterical disorders. It may even be said that if emotions, by weakening the critical sense, may prepare the mind to submit to suggestion, they nevertheless exclude suggestion for the time being, and when they are intense they prevent the development of pithiatic disorders, or even cause them to disappear. *When the human soul is shaken by a profound and sincere emotion, there is no room left in it for hysteria* (50).

"The foregoing description will enable the reader to understand how hysterical manifestations may vary in form and frequency according to circumstances, in marked contrast with the permanence and invariability of other mental diseases. This changeableness is explained by hysterical phenomena being a product of suggestion. If emotion, as was once supposed, is able to produce them, they ought to be almost as common nowadays as they were in the past, for life's tragedies have not ceased to cause moral upheavals.

"A new question arises. Do the affective states which we distinguished from emotion resemble emotion or differ from it in their relations with hysteria? Like emotion, they are incapable by themselves of

creating pithiatic phenomena, but unlike emotion which is incompatible with such symptoms they play an accessory part in their pathogeny, as we shall endeavour to prove.

"In this connection I must say that my interpretation of hysterical manifestations has been met by the following argument: Mere ideas cannot have sufficiently great influence on conduct or moral health to produce such disturbances.

"This objection which is of a speculative character cannot hold good against the facts which I have advanced, but even theoretically it does not appear to be justified, and I have endeavoured to refute it in a work written in collaboration with Dagnan-Bouveret entitled *Emotion and Hysteria* (59).

"It might have some significance if by suggestion was meant the influence of a purely abstract idea, if indeed such ideas possess any psychological reality. Numerous studies including those of Ribot have shown that all ideas, even the most abstract, possess a certain affective element, however weak it may be. Moreover, in the cases under consideration there is no question of ideas of this kind; we are dealing with ideas with an extremely rich affective content whose influence can certainly not be disregarded. I have often insisted on the fact that the psychical process of suggestion has an element of simulation, the degree of which varies with the individual and with the circumstances in which the hysterical phenomena appear. The latter, it may be said, are due to an unconscious or subconscious simulation, or a sort of demi-simulation. All these terms, which clearly indicate the mechanism on which the pathogeny of the pithiatic disorder depends, enable us to approximate it, at least in one of its aspects, to a voluntary act. This comparison will allow me to explain my meaning. If a voluntary act be considered, it is seen to be the realisation of an idea, of a project formed in advance, and in part at least, the result of an intellectual operation. But if the causes be investigated for adopting one idea in pre-

ference to another for carrying out this act, it will be found that the choice was determined by the sentiments to which it gave rise. In like manner the hysterical subject conceives the idea of a morbid condition, and realises it when this idea becomes implanted by its systematised affective elements, *i. e.* when it arouses in the patient the desire of certain advantages, or a continual uneasiness, or an abnormal desire to astonish or attract attention ; in short, creates the infinite variety of motives which act upon the easily influenced will of such patients. The rôle of the systematised affective elements is then clearly revealed ; they establish the idea and give it the power of a plastic realisation.

" To sum up, emotion (in the sense of a shock) cannot by itself cause the appearance of hysterical symptoms contrary to what was formerly admitted without dispute ; it is even opposed to their development and is not compatible with them. To enable them to appear the symptoms require the intervention of a suggested idea, which, it is true, is supported by systematised affective states."

Although the essential features of the conception which has just been described have been admitted by the majority of neurologists, some sides of the question have remained until recently the object of dispute, which has cropped up again in relation to the neurology of war. These are questions which we shall have to discuss in the following chapters.

CHAPTER II

HYSTERIA OR PITHIATISM IN THE NEUROLOGY OF WAR

Ever since the medical world became familiar with the influence of suggestion on the genesis of hysteria, whether the suggestion emanated from the family, the hospital or the doctor himself, and susceptible persons have been protected from its effects, the manifestations of hysteria have become much less frequent than formerly, especially in hospitals. Our observations, and those of Chauffard, Brissaud, Chaslin and many others bear witness to this. But as human nature has not changed, there was every reason for supposing that hysteria would increase under favourable conditions. Has the war produced such conditions, and are pithiatic phenomena frequently met with at present? Opinions on this question do not agree. Whereas Guillain (96), for instance, regards hysteria as very rare, Chavigny (99) considers it to be very common, but he thinks it is, as a rule, not recognised at the present time. "The exceptional circumstances of the war," he says, "have produced a real harvest of hystero-traumatic manifestations." Roussy (78), (79) estimates the proportion of hysterical manifestations to other nervous disorders from 10 to 15 per cent. Grasset's estimate is 42 per cent. (76). What are we to think in the presence of such divergences of opinion? It must undoubtedly be admitted that the diagnosis of hysteria has been improperly applied to entirely different nervous phenomena; it is none the less true that hysteria is often observed in the neurology of war. This is not a mere impression of ours; our opinion is

founded on the rapid disappearance of a large number of nervous symptoms which we obtained by various methods of counter-suggestion. This criterion is conclusive; as we have seen, it is an evidence of the pithiatic nature of the disorder in question.

In the cases with which we were concerned hysteria was either pure or associated with various organic disorders, and the latter form appeared to us to be much the commonest.

We will group the facts relating to the hysteria of war in two sections, entitled "Genesis" and "Symptomatology" respectively, and we will endeavour to draw suitable conclusions therefrom.

In our description of the various pithiatic phenomena which we have observed we shall have to insist on certain characteristics which enable them to be distinguished, but we shall not deal with the principal part of the diagnosis, especially that concerning paralysis, and contractures, until we have given a description of nervous troubles of a reflex character. We shall follow the same plan as regards treatment.

The Genesis of Hysterical Phenomena

What is the respective importance of emotion, traumatism and suggestion in the genesis of the hysterical phenomena observed in the neurology of war?

The present war which exceeds all others owing to the number of those engaged, the formidable character of the engines of destruction and its duration, presents a unique field of study for those desiring to determine the relative importance of the various provocative agents of hysteria. We may remark in passing that predisposition, personal or hereditary antecedents, the nature of the individual and the emotional constitution appear to be of secondary importance. The hysterical symptoms observed at the present time, as Grasset remarks, appear to develop chiefly as the result of moral and physical strain, and of commotions which diminish the psychical resistance of the individual and

predispose him to nervous disorders of this kind, however insusceptible he may appear.

We may mention that one of us as long ago as 1892 in a study of hysteria and hystero-organic associations wrote as follows (19): "It is undoubtedly one of the most widespread affections, and for my part I am inclined to believe that there are very few individuals who escape when placed in certain circumstances, and under the influence of more or less active occasional causes.

"This proposition, I hasten to say, applies to hysteria minor only; I believe, in fact, that hysteria major hardly ever develops except in individuals who are predisposed to it by heredity or nervous antecedents, and that it is the appanage of a neuropathic aristocracy; whereas hysteria minor is, so to speak, open to every one."

Rôle of Emotion

Some neurologists attached to sanitary units behind the lines thought that they could prove by their observations that emotion alone could cause hysterical manifestations to the exclusion of any factor of "suggestion." But to prove their point they appealed to the statements and explanations made by the wounded themselves. This examination by "retrospection" (*v.* p. 427) is not conclusive, as we have shown, being open to many sources of error. It is essential to depend entirely on examination by "prospection" (*v.* p. 427), as was done by our colleagues at the front who were able to judge of the effects of violent emotions on the spot. How do they enlighten us in this respect?

"I was able to follow a bombardment carefully," said M. Dide, chief medical officer to the 24th Battalion of "Chasseurs Alpins" (101). "My aid post was situated about five hundred yards from the first line on the day of an attack. The bombardment of the German counter-attack was chiefly directed against our second line, and the

communication trench from my aid post to the trench suffered especially. The two companies who were collected there to support the attack, if required, were kept standing still, and were subjected to an accurate fire by heavy artillery. The losses were high, since we had to remove thirty-two dead bodies which had been almost blown to pieces as well as the wounded. After two hours of this experience some movement took place. It was thought that the order had been given to retire, and I saw half a company making their way to my aid post in a bewildered condition just as if they had been surprised in their sleep. I gave them a few words of encouragement, and, new orders having meanwhile arrived, they returned to the firing-line in the most perfect order. I have referred to this episode not as something exceptional, but as one which happened to come under my personal observation. It appeared to combine all the circumstances likely to cause nervous disturbances, viz. the extreme violence of the bombardment (several explosions a second), the heavy losses in killed, the appalling spectacle of the mutilated bodies and the absence of anything to distract attention, occurring as it did to troops standing still and waiting to fight.

"Neither on the day of this sanguinary affair, nor in the following weeks, was there a single case of nervous disorder as the result of the explosions.

"During a whole year I observed only a single case of delirium, oneiric in character, following the explosion of a trench mortar.

"I attribute the extreme rarity of the nervous disorders following explosions to the excellent *moral* of the troops. In any case, it must be admitted that, though I had a better opportunity of detecting such disorders than many medical officers, I looked for them in vain. . . . The extreme rarity of functional motor disorders in active units under energetic leaderships," adds Dide, "ought to be widely known. In the course of a year, in spite of the incessant activity of the unit to which I am attached, I saw only a single case of functional paralysis following a 'period of meditation' for a week after a shell had exploded close at hand. I had the opportunity of observing thousands of

men; and we had to suffer everything that war can invent in the way of producing emotion and anguish. . . .

"Functional disorders, in my opinion, can occur only in individuals whose emotional tone has relaxed. In a glorious unit like my own the war proved to me that neither privation nor formidable and incessant emotions could cause these phenomena in the immense majority of men whose strength was derived from the belief that they could challenge any one in bravery."

Here is another important piece of evidence which gives us very definite information as to the effects of fear.

"After living," write du Roselle and Oberthur (101), "in a sector where bombardment by large shells, mines and trench engines of all kinds was particularly continuous and energetic, we had set up at a very short distance from the trenches a fairly large aid post with beds in which it was possible for us to keep a few wounded under observation for the time required. We made a special point of noting the pathological phenomena following explosions as soon as they actually occurred. We are of opinion that some account of these daily observations made during action may be useful, though they were necessarily rapid and incomplete in consequence of the insufficiency of our methods of investigation.

"Whether due to the explosion close at hand of a mine or torpedo or an ordinary 77, or the whistling of bullets, or the spectacle of a scene of carnage, the state of emotional shock in the course of battle remains much the same. *There is no emotional reaction peculiar to large projectiles.* Whatever may be the cause, the individual will react in his own manner, and according to his own fancy: the post-emotional state thus produced will be, properly speaking, one of the infinitely varied pathological expressions of fear, such as cries, tremors, tears, and rushing forwards accompanied by various psychical manifestations and convulsive attacks; in short, by apparently hysterical phenomena.

"We may remark in passing that the convulsive attacks

which occur under such circumstances with or without rushing forwards, and often labelled unreasonably 'epileptic attacks' or 'epileptiform attacks,' very exceptionally occur in an exposed position; it is always at the bottom of a trench, in a shelter, and often also at the beginning of a bombardment, or on going up to the trenches; we never saw a case among men who were going to be relieved. When they rush forward it is very rarely in the enemy's direction, and almost invariably the patients come without hesitation to an aid post or a company headquarters.

"These men with their haggard and hallucinated looks, who make no reply to questions and do not recognise their officers or fellow-men, retain a lively instinct of self-preservation in their subconscious mind."

This interpretation may be illustrated by another example.

"One day during a violent bombardment, some men suffering from 'commotion' came helter-skelter to the aid post. They had no obvious wounds, but came tottering along with a typically haggard expression apparently unable to respond to questions or to recognise any one they met. One of us walked up to the soldier who seemed to be the worst sufferer and tried without success to show who he was; finally the man was dragged to the aid post by the arm so that he could be examined at leisure. Just as he was going in he saw the recent signs of a shell which had penetrated through the top of the entrance. The man at once stepped back, struggled and cried out, 'No, major, I won't go in there, shells were falling there too, not long ago.' It was impossible to make him advance a step. As soon as he was let go, he ran with his comrades in misfortune to hide himself in a little dug-out opposite, which we had made to protect the wounded until they could be evacuated. The men remained crouching there, trembling all over, not answering any questions, and apparently incapable of any voluntary act. But when the ambulances came to take them away there was no need to give them any explanation; they quickly understood, and took their place without any

help in the most normal manner possible. Their journey was quite uneventful."

Clovis Vincent, who remained a long while in the firing-line, where he distinguished himself by his courage and sang-froid, made some interesting observations as to the effects of emotion. We will quote the following extract from the notes which he sent us—

"In places where projectiles came in large numbers causing deaths and wounds I never saw any hysterical phenomena such as fits, contractures, or paralysis of one or more limbs. I saw emotional phenomena only, of which the most frequent were tears, trembling, a state of hebetude in which the man does not move or answer questions, tachycardia, tachypnœa, and micturition."

To this evidence we must add the series of striking observations made by Clunet at the time of the recent shipwreck of the mail steamer *La Provence II*, on which he happened to be. The following is an extract from the note which he kindly sent us.

"When *La Provence II* was torpedoed on February 26, 1916, we were able to study the manifestations of emotion close at hand immediately after the accident and *apart from any commotional state.*

"We found that the pithiatic phenomena did not occur till later, when the survivors were in safety. These phenomena yielded to an energetic treatment which was immediately applied, and did not recur during the week which followed the accident. To make our description clearer, we will divide our experience into four periods.

"1. On board the boat, during the seventeen minutes between the explosion of the torpedo and the complete disappearance of the boat (period of emotion pure and simple).

"2. In the sea for eighteen hours, including a whole night hanging on to a raft (period of emotion and physical suffering due to cold).

"3. On board the torpedo-boat which came to our assistance the next day (period of hysterical phenomena, and treatment).

"4. Off Milo, on board different vessels among which we were distributed, during the week preceding our departure (period of return to normal life).

"1. *On board. Period of emotion pure and simple.*—At 3 p.m. I was just going to read in the saloon when I heard a faint explosion (like that produced by a 77 three hundred yards off), and I felt a slight quivering of the ironwork, as if the boat was running on a rock, and the machinery stopped immediately. I went up on the bridge and found the ladders filled with a dumb and anxious crowd. On the bridge I met a naval officer who was a passenger, wearing his lifebelt. He was calm but quite livid, and said, 'We've been torpedoed; it's all up.' I tried then to get to my cabin to find my money; it was impossible, ladders and corridors were completely blocked by the soldiers; the news had spread, 'We've been torpedoed.' The panic began about five minutes after the torpedoing, when the boat, which was just beginning to sink at the stern, seemed motionless, and looked as if she could weather the shock. The panic remained a silent one. There were no cries, only a few gasps from men who were being choked or stifled in the crowd on the ladder.

"Some were in a state of stupor, and remained motionless on the bridge, but the majority were in a state of great agitation and acted in an irrational way; some crowded into the boats on the bridge without trying to lower them, others ran up into the rigging, while others fired their revolvers or rifles into the air. I went up to a ship's officer who with a fixed expression was discharging his revolver into the sea and asked him if he had any orders. He seemed to wake up out of a dream, and replied, 'Orders, why should I? It's all up.' 'Then why are you firing your revolver?' 'To stop the panic.'

"In a few moments the firing took a different turn: an infantry officer shot himself in the head. This example was soon followed by several men, and I witnessed a real epidemic of suicide. Many men threw themselves into the

sea from the bridge in the front part of the vessel, which was now raised more than fifty feet above the surface of the water.

"In this panic-stricken crowd there was not a single pithiatic phenomenon such as fits, convulsions, paralyses or barking—almost all kept silence; but there was no mutism, they replied quite well to a direct and definite question. Side by side with these panic-stricken people, a few officers, soldiers and sailors maintained complete self-possession, endeavouring, but unsuccessfully, to launch the boats into which the men had crowded, and throwing everything that could float into the water.

"At 3.15 p.m. the stern began to sink, so to escape the fall of the masts and the suction cone produced by the sinking ship, I made my way to the level of the water towards the stern and swam to a small raft which was floating a few hundred yards from the vessel. At 3.17 p.m. (in spite of being in the water my watch had not stopped) there was nothing left on the water but wreckage, swimmers and drowned.

"2. *On the raft. Period of emotion and physical suffering due to cold.*—I was the first to reach the raft, where I was soon joined by two other swimmers; we at once endeavoured to save as many of our comrades as possible, so that in a few minutes there were seventeen hanging on to the open raft measuring less than ten feet by four. It was very cold (not more than 44–46° F.), the water seemed warmer, but we were covered with water by every wave, and the maceration of the skin soon became very unpleasant.

"Several of our companions had stripped so as to swim better, and we shared our clothes with them.

"At first there were isolated expressions of despair and lack of discipline—I was the only officer in the little group—some crying out, 'We are lost; it's sad to die like this.' In about an hour's time things improved, and the men recovered courage. They followed the lead of a cheerful fellow, who had often been 'in a much worse hole' and had always got out of it, and was sure that he would this time. I dressed a broken nose and collected objects

floating in the sea, *e. g.* some apples, a piece of bread, a flask half full of wine, some Cosson signals and an oar. On my advice the food was kept in reserve for the second twenty-four hours. We changed places frequently to prevent our joints getting stiff, those in the centre of the raft who did not have to hang on so vigorously as the others rubbed their neighbours and smacked them on the back.

"In the middle of the night a native of Martinique said that he was done for; he began a religious lamentation, thought he saw heaven, commended his soul to the Virgin and the saints and began to cry. His neighbours appeared to be impressed, so I told him to say his prayers to himself. As he did not obey, I took him by the wrists and threatened to throw him into the water, which had the effect of keeping him quiet. The *moral* which had been shaken for a moment by this occurrence was re-established by a funny yarn about Morocco, which set every one laughing. During the eighteen hours on the raft I did not see any pithiatic phenomenon. None of my men (sailors, stokers and colonials) died, and none of them showed any nervous disorders during the following days.

"3. *On the torpedo-boat. Period of pithiatic phenomena and treatment.*—The rescuing began at about 7 a.m. and was extremely long and difficult owing to a heavy swell. We were all taken off at about 9 o'clock. I went to see the survivors collected in front of us. Some of them were wounded (from struggling during the last moments on the *Provence*, or from the fall of rigging). One even had a fractured thigh, and I rigged up a make-shift splint for him. Several showed neuropathic or pithiatic phenomena, such as quadriplegia, paraplegia, or mutism, incessant snarling, spasmodic weeping, groaning, hiccoughing, barking and shaking amounting to spasmodic movements of the upper limbs, but no generalised convulsions. I sent the patients down to the engine-room close to the engines, where the temperature was very high. The number of my patients increased as new survivors arrived, so that out of six hundred shipwrecked persons collected by our torpedo-boat, I found thirty to forty with nervous disturbances.

The treatment was simple, they were entirely stripped in an over-heated room, and energetically rubbed by two vigorous sailors with a hair glove soaked in alcohol. As soon as they had been warmed externally and internally (rum), I took each one separately and smacked him harder and harder until the disturbances disappeared, all the time speaking kindly to them and expressing my delight at the rapidity of their recovery. No one resisted more than ten minutes; many were cured by contagion, on witnessing the treatment of the others.

"The majority expressed to me their gratitude on the spot and during the following days.

"In my opinion the cold, which caused a large number of deaths on many of the rafts, played a part in the phenomena of contracture which in many cases were sufficiently intense to prevent the appearance of the slightest tendon reflex in the affected limbs. In one case of contracture in the lower limbs, the plantar reflex was impossible to obtain for many minutes, and then it became normal.

"4. *Off Milo. Period of return to normal life.*—I stayed at Milo about a week before returning home. During this time I had several opportunities of seeing my patients on the torpedo-boat, but none of them showed any marked symptom of nervous disorder. It is probable that if at the time when their neuropathic symptoms appeared these persons had been put to bed and received delicate attention from admiring women, the therapeutic results would have been very different."

As Clunet remarks, the observations on board *La Provence II* in the first stage of the catastrophe are particularly interesting to one who wishes to judge of the rôle of emotion in the genesis of nervous symptoms. Emotion in the sense of a shock must have reached its maximum intensity, and was acting without any physical commotion being superimposed on the psychical shock; its field of action was thus much easier to define. Now, in the midst of all the mental disturbances which arose under these circumstances, no pithiatic disorder appeared at that time. We may

add that one of us served in a divisional ambulance during the first eight months of the campaign. All the wounded and sick from the corresponding section passed through this clearing-station, which was operating in a zone from two to three miles behind the firing-line, first in Alsace, then in the neighbourhood of Saint-Dié, and lastly on the Somme. Neither in the military nor the civil element did he meet with any cases of hysteria, either during the period of retirement or at the time of the exodus of the population which, alarmed by the first effects of the bombardment of Saint-Dié, made a hasty departure, and for several hours defiled in front of the ambulance. Nor did he see any when the ambulance was bombarded in turn.

The last circumstances deserves to be related in some detail, for it may have seemed *a priori* specially suitable for the development of hysterical symptoms. It was on August 28, 1914, when the ambulances 1/14 and 3/14 had just been drawn up side by side by the Burgonce after a period of ten days' continuous retirement which had had a powerful demoralising effect upon the men. All of a sudden heavy shells, which had hitherto been falling at about a mile off, began to fall nearer and ended by reaching the ambulance. Although the bombardment lasted only an hour, out of an effective of forty men one of the ambulances had seven wounded and twelve killed. The latter were hardly recognisable, in many of them the trunk had been separated from the limbs, which had been torn off and hurled to a distance. The effects of the bombardment had made a great impression upon the survivors, all the more so as they were not combatants but hospital orderlies, to whom this painful episode was unexpected, and constituted their baptism of fire. And yet, neither that day nor during the following weeks did any of them present any hysterical manifestation.

He had, however, the opportunity of seeing two individuals with hysterical symptoms, but this was

during a relatively calm period, among troops in a rest camp.

In a communication made with Major Plisson to a medical meeting of the 2nd Army in March 1915 he drew the attention of his colleagues to the extreme rarity of hysterical accidents seen at the front, showing the similarity of this observation with those made before the war (*e. g.*, in hospitals, or the enquiry into the earthquake at Messina, *v.* p.429).

This communication did not provoke any contradiction.

It is important to note that the evidence we have brought forward has met with no opposition. At the front, therefore, under conditions likely to cause the most violent emotions various emotional disturbances may be observed, but no hysterical symptoms have been seen to develop on the occasion of an emotion.[1]

The results of the observations have been quite different in places distant from the firing-line. At nine to twelve miles from the front lines in the sanitary units in the line of communication hysterical manifestations are frequent. This fact has been the motive for creating neurological centres at the front for the special purpose of retaining persons suffering from disorders of this kind in order to submit them immediately to psychotherapy, which is all the more likely to be successful the less it is delayed.

Pithiatic phenomena are still more frequent in neurological centres at the base among soldiers who have spent many months in various hospital units, but their number varies considerably comparing one centre with another. Considering the time of onset of these phenomena one is not justified in eliminating suggestion, which is all the more likely to intervene

[1] Information supplied by other medical officers at the front with whom we had the opportunity of discussing this question has been entirely unanimous : *e. g.*, Major L. Lévy and O. Monod, who were attached to ambulances engaged near the firing-line, assured us that they had never seen any symptoms resembling hysteria.

as men suffering from severe emotions often show an abnormal degree of suggestibility associated with a slight confusional syndrome. This fact has been brought into prominence by George Dumas, psychiatric expert in the 1st Army, in a very remarkable analysis of the mental state in emotion (101).

" All persons experiencing a great emotion and all who have received a commotion, or almost all, present characteristic mental disorders, when an examination is made of their intelligence, memory or imagination. In some these disorders are very marked, and one finds dulness, dysmnesia of evocation, and especially of fixation, and fairly often oneiric delirium, in other words, very definite and pronounced symptoms of mental confusion.

" In others one finds the same symptoms much less marked and usually reduced to a certain difficulty in comprehension and fixation; one is thus led to conclude that the confusional syndrome exists in various degrees in most of those suffering from great emotions or commotions reaching us from the front. . . .

" These observations are not only of interest in themselves, but they are of still greater interest when an attempt is made to find an explanation in their state of mind for the nervous disorders which these patients frequently present. The pithiatic character of a very large number of symptoms is not contestable, and has not been contested, and their pathogeny has almost always appeared to me to be associated with a slight confusional state. This condition, the mental characteristics of which I have described, is revealed by a special suggestibility, an absence of initiative, and a tendency to exaggerate all the difficulties of an effort and to transform them into permanent obstacles. The patient who has not lost consciousness of himself, or who is beginning to find it again, readily carries out a negative auto-suggestion in the performance of his various functions, and in the presence of the slightest obstacle he imagines that he is dumb, paralysed or deaf. . . .

" In offering an explanation of the hysterical symptoms of our patients one should admit the existence of an inter-

mediate period which is interposed between the initial shock and the appearance of the symptoms, this intermediate period being one of slight mental confusion. This allows the patient the exercise of a functional activity during which he easily runs the risk of giving way to auto-suggestion. This intermediate confusional state may be suspected or even found in many of the cases published a long time ago, in which the authors attached importance only to emotion, the nervous shock and the symptoms which followed. There is a chance that in some cases the confusional state may have passed unnoticed."

Violent emotions obviously prepare the soil and create a predisposition for hysterical manifestations. They increase suggestibility at the expense of the critical sense, and by occasionally producing actual states of slight mental confusion. The events of the war have confirmed in this respect the following opinion expressed by one of us in the course of a discussion on emotion (55). "I readily admit that a moral upset by weakening the critical sense may increase the patient's suggestibility"; and Dupré said in the course of the same discussion, "Emotion being an element in the dislocation of the personality favours suggestion." But the hysterical manifestations which arise from this new state of mind do not appear at the moment of the emotional shock. Striking observations such as we have just related confirm this opinion, which we had already arrived at from observations made before the war. Between the emotional shock and the appearance of pithiatic phenomena there is a fairly long interval to which Charcot gave the name of "period of meditation," during which auto-suggestion and hetero-suggestion have plenty of time to intervene. In support of the contrary idea according to which hysterical symptoms could be caused by emotion exclusively, the only cases worth considering are those in which the disorder had immediately succeeded the moral shock.

The study of hysterical symptoms observed in the

neurology of war has not furnished any conclusive case of the kind.

As regards the opinion of Dejerine (55), who formerly used to attribute the absence of neuropathic phenomena in soldiers who had faced death and seen their comrades fall at their side to the nature of the emotion which was, so to speak, foreseen, such a view in our opinion seems hardly tenable. Considering the range of heavy artillery and the power of big shells which come from every conceivable direction and unexpectedly explode sometimes in the midst of a rest camp with results comparable to an explosion in a coal mine, how would it be possible to admit that the resulting emotion does not constitute an emotional shock in the most complete acceptation of the term ?

We have even maintained formerly that hysterical symptoms disappeared infallibly under the influence of a profound emotion.

In the publications of these last two years we have found only a single case, recorded by Chartier (90), which appeared to be in opposition to this point of view. His case was that of a man with paralysis of the hand without any sign revealing the existence of a lesion of the nerve centres or of the peripheral nerves, and which the author attributed to hysteria. Under dramatic circumstances and at the moment when the wounded man, who had already tried to commit suicide by throwing himself into the water, was in danger of death, Chartier found that the paralysed hand did not show any movement. This case would be conclusive if the diagnosis of hysteria was incontestable, but this was by no means certain. It should be noted that this paralysis developed after a gunshot wound, was accompanied by a slight degree of contracture, and that the results of psychotherapy were very incomplete. For all these reasons one is justified in thinking that it was really a paralysis with reflex contracture. Now, disorders of this kind are hardly more amenable to psychic influences than those which are connected with lesions of the nerve

centres or nerve trunks. None of the features of the case allow one to exclude this diagnosis, which was not even considered. No fact, therefore, has hitherto been brought forward to contradict what we said, viz. that hysterical symptoms and violent emotions are incompatible.

Rôle of Traumatism and the Doctrine of Hystero-Traumatism

In 1886 Charcot drew attention to the rôle of traumatism in the genesis of local hysteria; this conception was defended and developed by his pupils, especially by Bouveret (13), and by Blum (23). Until then nervous troubles of this kind (railway spine, railway brain) were considered without any discussion as organic manifestations, in conformity with the opinion expressed by Erichsen in 1866.

The idea of hystero-traumatism was a very fruitful one, and recent events have shown the frequency with which traumatism determines the outbreak of pithiatic phenomena. It is equally true that nervous symptoms of an entirely different nature have very often been attributed to hystero-traumatism. Thus at the beginning of the war medical men seemed to have completely forgotten the existence of those amyotrophic paralyses and so-called reflex contractures which have been so well described by Charcot and Vulpian. Any paralysis or contracture which did not present any of the signs of the classical organic affections were attributed without hesitation to hystero-traumatism. All the cases, therefore, which have been published under this name cannot be accepted. This is a point to which we must often return later.

We will now discuss by what mechanism traumatism and especially the traumatism of war can produce hysterical symptoms.

In the case of local traumatism (gunshot wound, shell wound or contusion) the question is relatively simple; the pain and the more or less persistent

inhibition of movement resulting from it are the starting-point of a process of auto-suggestion. The organic factor, however slight it may be, acts as a bait, so to speak, for the hysterical symptoms which settle in the region of the trauma and usually survive the exciting cause.

In the case of another variety of traumatism consisting in commotions caused by explosion of large shells, minenwerfer and mines the question is more complicated. First of all the actual nature of the nervous troubles which develop in such cases may be a matter for discussion (101). Whereas Roussy, for example, thinks that it is exceptional for explosions a long way off to produce organic lesions, Guillain, on the contrary, maintains that organic disturbance of the neural axis in such cases is frequent. The former is of opinion that the nervous troubles which result "should in the immense majority of cases be classified among the so-called hysterical or pithiatic disorders"; while the latter, in the course of the discussion at the Neurological Society devoted to the study of commotion due to shell explosions, expressed the following opinion: "Many patients are wrongly considered as pithiatic, hysterical, exaggerators or simulators."

Between these two extreme opinions is to be placed that of Claude: "What we most frequently observe in the zone of the interior are psychoneuropathic states, focal organic lesions constituting the exception, but owing to the frequency of changes in the cerebro-spinal fluid and especially of variations in its tension, it is possible that the functional disorders observed are kept up by slight modifications in the composition and circulation of the cerebro-spinal fluid."

Hystero-organic associations appear to us to be common after traumatism of this kind. The reality of an organic lesion sometimes admits of no discussion, even in cases in which the remarkable success of counter-suggestion affords a proof of the pithiatic nature of the nervous disorders observed. This fact is illustrated by the following case (Leriche and Froment)—

As the result of the commotion produced by the explosion of an aerial torpedo a soldier fell and lost consciousness. He was taken in a comatose state to the ambulance and thence to a clearing-station. The coma ceased in forty-eight hours, but complete mutism and contracture of the lower limbs developed. Lumbar puncture gave issue to a definitely hæmorrhagic fluid. On close examination, however, the contracture presented all the characters of a psychical contracture; in a few moments it could be caused to disappear by counter-suggestion and the patient was enabled to walk. During a second spinal puncture he was seized with a spasm of the upper limb, which was also caused to disappear at once by persuasion. In the night following this examination the patient began to speak in the course of a dream, and from that moment he was cured of his mutism. A month later he was transferred to a neurological centre, where he was again examined by one of us for motor troubles resembling rhythmic chorea, which rapidly subsided when he was assured that the symptoms were not serious. In a few days he showed some impatience to leave hospital, declaring that he was perfectly cured and asking to return to the front. It is interesting to note that he was a vigorous, well-balanced individual without any neuropathic taint and not alcoholic. The pithiatic nature of the symptoms was so obvious and indisputable that had it not been for a lumbar puncture, one would have had no hesitation in admitting that it was a case of pure hysteria. The result of the examination, however, enabled us to affirm the reality of a central hæmorrhage. In other cases there are no grounds for supposing the existence of a concomitant lesion, and traumatism alone appears to have caused the pithiatic disorders. Whether the commotion caused by a shell explosion can produce lesions of the nervous system or not, there is every reason for supposing that it can increase the suggestibility and be thus the cause of hysterical disorders as much as a psychical commotion or emotion.

The hysterical symptoms will vary in their form, intensity and localisation according to the sensations caused by the actual shock and the various circumstances in which the patients are placed. But it is essential, we repeat—and the reader will better appreciate the justness of this remark after reading the chapters devoted to reflex nervous phenomena—not to attribute to hysteria all the disorders following traumatism and independent of any lesion of the central or peripheral nervous system. However correct may be the idea of hystero-traumatism, it has sometimes been misapplied in the neurology of war.

Rôle of suggestion.—We may remind the reader that, according to our conception, suggestion plays the essential part in the production of hysterical manifestations : these are absent or very rare in circumstances unfavourable for suggestion; they are very common under the opposite circumstances, their frequency, intensity and duration depending on the conditions which are suitable to produce and maintain suggestion. Some neurologists, however, thought they had the right to assert that in certain cases of hysteria observed in the war suggestion could not have intervened. To prove this they thought it sufficient to mention the absence of any previous medical examination, and to declare that they had taken all the requisite precautions for avoiding medical suggestion. But the defect of such a method of reasoning is that it does not take into account other modes of hystero-suggestion and auto-suggestion. It must be admitted that under the present circumstances, considering what they see and have heard said or think they know of the effect of various wounds in war, the patients concerned are almost inevitably exposed to suggestions of all kinds, of which they may not be conscious, but which, though unrecognised, are none the less efficacious.

The result of the evidence set forth in this chapter has been that the neurology of war has not produced any argument against the opinion which we have

maintained. It even tends to strengthen the idea of the indispensable rôle of suggestion which determines the appearance of hysterical phenomena, moulds them, and, as we shall see, guides their course. Emotion and traumatism only predispose to manifestations of this kind. They increase suggestibility and sometimes provide the elements of an auto-suggestion.

CHAPTER III

SYMPTOMATOLOGY

Although hysterical manifestations are common in the neurology of war, it is equally true that other nervous symptoms besides pithiatic phenomena are constantly described by this name, although they do not originate in suggestion and counter-suggestion cannot disperse them.

This confusion is much to be regretted, inasmuch as it involves two kinds of disorders quite distinct in their mode of evolution, prognosis and treatment, as well as in the medico-military decisions they give rise to.

We will therefore endeavour to separate what really belongs to hysteria from that which has been improperly attributed to it, by a critical examination of cases published during the two years of war.

Hysterical manifestations are essentially polymorphous. As they are the product of suggestion, they are modified by current ideas and are modelled on the conceptions of the individual.

A complete and detailed description of the hysterical symptoms observed in the neurology of war would consequently present no special interest. We will merely allude to them, laying special emphasis on those which are most frequent, with practical suggestions in passing, and dealing particularly with questions which, still forming the object of controversy, deserve to be discussed.

Hysterical Stigmata

Are the observations which have been made during the war likely to make us resume the old conception of the permanent stigmata of hysteria ?

A few neurologists seem to be inclined to this opinion. Lentz (66) has noted the existence of a large number of hysterical stigmata in the soldiers of a Russian regiment which had taken part in many battles and had suffered considerable losses.

Mondino (101) also remarks that in numerous cases following explosions " a persevering physical examination " had always enabled him to find undoubted hysterical stigmata.

The mere use of the term " persevering " applied to the examination of subjective phenomena justifies one in thinking that medical suggestion intervened in these investigations.

Chavigny and Sollier have, it is true, found stigmata in subjects who had undergone no previous examination, and although they had taken every precaution to avoid medical suggestion, but this opinion is in opposition to that of a very large number of neurologists who are more convinced than ever by their experience in war that there is no foundation for a belief in the permanent stigmata of hysteria. As regards the so-called ocular stigmata, the reader will see later, in the paragraph on ' Hysterical visual disorders," what view should be adopted (*v.* p. 474). The nervous disorders, therefore, which have been described by the name of stigmata show a very variable frequency according to the observer, the cases observed, and the actual circumstances of the observation. In any case it is beyond dispute that at the present time these stigmata are infinitely rarer than formerly, and that medical men are more on their guard and so create them less frequently But it is natural that they should reappear as soon as proper precautions during investigation are neglected. Moreover, in order to conclude that these stigmata do not originate in suggestion it is not sufficient to eliminate medical suggestion only. It must not be forgotten that other forms of hetero-suggestion or auto-suggestion may still intervene. We have already dwelt at sufficient length on this subject.

What clinical significance should be given to stigmata? They have exactly the same meaning and value as all the other symptoms which suggestion can produce. They constitute in themselves hysterical phenomena (we have never disputed this), but—and this is the essential point on which we must insist—they cannot in any case justify us in attributing to hysteria the nervous symptoms which accompany them (*v.* p 415).

Some join Chavigny (99) in thinking that by refusing these stigmata the position they formerly possessed there is a risk of hysteria being overlooked. Our opinion is that it is much more dangerous to attach an excessive importance to these phenomena. In fact, by adopting this method in cases with hystero-organic associations which are so common in the neurology of war, there is a risk of not seeing the organic element. There is also another risk, viz. that of failing to recognise the numerous cases of paralysis and contracture in which stigmata are completely absent.

In the chapter on "Diagnosis" we shall see what method should be adopted and what other criteria should be taken to establish with all possible rigour the diagnosis of pure or associated hysteria.

Hysterical Fits

Hysterical fits have frequently been observed in some neurological centres.

They are distinguished from epileptic fits in the first place by the constant absence of objective signs characteristic of epilepsy: cyanosis of the face with lividity of the lips, subconjunctival hæmorrhages and subcutaneous petechiæ, and extensor plantar reflex.

The other characters of the epileptic attack—initial cry, sudden fall, complete loss of consciousness, biting of the tongue, presence of bloody froth on the lips, incontinence of urine, and subsequent prostration—though they may be valuable elements in diagnosis,

are not conclusive, for they may be consciously or unconsciously imitated.

Hysterical attacks are also distinguished by the form which they assume (wide-ranged movements, gesticulations and opisthotonos) (*v.* p.408), and by another character which is conclusive, the possibility of reproducing it exactly by suggestion, and causing it to disappear immediately by counter-suggestion.

This test is of great practical value when one cannot witness a spontaneous attack. We have very often had recourse to it with success. This is how we proceed—

We first of all announce in the patient's hearing that we are going to try to reproduce the attack, this being indispensable to enable us to lay down the treatment and to get a proper understanding of the case. We add that electricity is almost infallible in such cases, especially when the electrodes of the faradic machine are placed on a definite spot. It is not necessary to employ strong currents; sometimes, even, we content ourselves with merely applying the electrodes without passing the current.

Shortly after we have put the electrodes in position, we declare that the attack is going to begin and that the patient will struggle. To prevent his falling we put him at full length on the ground or on a bed. As a rule, in cases of hysteria the effects of suggestion appear rapidly: the legs become stiff, the body is thrown into contortions, and the fit proceeds with special modifications according to the individual. As soon as the attack is fully developed, we declare that we have an infallible method for arresting it. We then apply the electrodes to another region, or we exercise a pressure on the abdomen or trunk, saying in a confident tone that the attack will be over in a few moments. In fact, an hysterical attack produced in this way does stop, and the subject is at once able to answer questions put to him and to leave the room where the examination has taken place.

It must not, however, be forgotten that the same

individual may be subject sometimes to hysterical fits and sometimes to epileptic fits; it is therefore advisable to question the attendant or friends to ascertain whether the spontaneous attacks closely resemble the artificial one. If this is the case, no further doubt can be entertained as to the hysterical nature of the attacks.

Finally, there is one other remark which we should make: the patient during his attack of hysteria and while apparently quite lost to all that is happening about him hears everything that is said, as in his normal state. It is therefore essential to avoid any remarks which might originate a suggestion.

Disturbance of Sensibility

Hysterical anæsthesia.—Hysterical anæsthesia appears in various guises and does not obey definite rules.

It sometimes assumes the form of hemianæsthesia, as we have stated in dealing with the stigmata, sometimes the segmentary form, and sometimes the form of isolated patches; not unfrequently it imitates the anæsthesia of peripheral type so frequently observed at the present time, but in that case it reproduces only approximately the anæsthesia associated with lesions of the nerve trunks.

These sensory disturbances do not cause any inconvenience; the patient is never known to burn himself like a wounded man suffering from anæsthesia due to a lesion of the ulnar or median. They often vary from one moment to another, and become modified according to the conscious or unconscious suggestions of the observer: they diminish and tend to disappear as soon as no further notice is taken of them.

The following, in our opinion, is the right way to proceed in an examination of the sensibility, which is always rather a delicate matter. First of all it is advisable to blindfold the patient and carefully to avoid influencing him by the questions put to him. One must also take care not to ask him if he feels as

distinctly in one region as in another, and one should refrain from any reflection which might suggest to him the idea that his sensibility might be diminished. The best thing is merely to say before beginning : " Attend to me, and tell me as soon as you feel that I am touching you." We often appeal to the patient's self-esteem, by telling the students loud enough for him to hear that, judging by his looks, he must be an intelligent person and will probably give us exact information. We then proceed to touch very lightly different parts of the body, pinch and prick the skin, exercise pressure upon it with the finger, flex and extend different segments of the limbs, separate the patient's fingers and cross one over the other, give him various things to handle of a round, cubical, cylindrical, spherical or oval shape, and apply warm or cold objects to the skin. All this is done sometimes on the left and sometimes on the right, sometimes at the extremity and sometimes at the base of each limb, without the examination seeming to follow any preconceived idea or any apparent rule. If the subject does not say spontaneously what he feels, we simply ask him : " What do you feel now ? What am I doing ? " It is most important, to try to bewilder the patient, and to act as if one wanted to catch him in the act of contradiction, without this method of procedure in any way implying a suspicion of simulation. One should also carefully note all the movements and gestures caused by suddenly pricking or pinching a region supposed to be anæsthetic, or by the unexpected application of a hot or cold object. When these precautions are taken to avoid any suggestion and any wilful or unconscious trickery, one becomes convinced of the variety and instability of hysterical anæsthesia.

Lasègue, in an unpublished note which we owe to his grandson Cesbron (54), expressed himself as follows : " It is common to find that hysterical patients who have not yet been enlightened by the doctor's investigations do not make any mention of anæsthesia." " I

have carefully examined," he adds, "a large number of hysterical subjects and earnestly implored them not to omit any of the inconveniences they felt, and I have never met with a single one who spontaneously mentioned anæsthesia among the symptoms of which he had to complain. It is very different with patients whose attention has been directed to this kind of phenomena. But even when they have received this information, how many are there who really suffer from it or complain of any inconvenience resulting from their lack of sensibility?"

It is certainly due to the precautions taken that we have never found in the neurology of war a characteristic and stable hysterical anæsthesia, any more than we have met with stigmata.

Chavigny (99) declares, on the other hand, that such disorders are very common at present, but are usually overlooked. The article in which he maintained this opinion has been criticised as follows by A. Léri (104)—

"It is not without surprise that we have seen some of the best-known writers broaching reactionary theories with regard to the present conception of hysteria. According to them, during this harvest of hystero-traumatic disorders which we see to-day, there are numerous observers who would deny the existence of hysterical hemianæsthesia limited to one limb and other disturbances of the same kind; these are 'ignored diseases'; hysterical patients are systematically regarded as simulators or considered to be suffering from organic diseases; hysteria is actually an archaic syndrome which has become unfashionable and out of date; nobody dares to make a diagnosis of hysterical disorders, and there is no longer any question of it either in symptomatology or differential diagnosis.

"I suppose that this is a severe criticism of the partisans of the present conception of hysteria, who include to-day the overwhelming majority of the leading neurologists; but I am quite certain that this criticism is not justified. Unless I am mistaken, no

one has denied hysterical anæsthesia or deliberately regarded hemilateral or segmental anæsthesia which does not prove to be organic as signs of simulation; nor has any one maintained that the doctor alone was responsible for hysterical disorders, and that subjects who had not been subjected to any previous examination could not present perfectly genuine symptoms.

"The 'current doctrine,' which chiefly originates from Babinski's work, far from denying the existence of hysterical disorders, considers them, on the contrary, as the result of auto- or hetero-suggestion. They can merely reproduce, it is true, what the will can produce, but that by no means signifies that they are actually produced by the will, and that there is consequently any question of simulation. There is no doubt that they can be caused by a medical suggestion, but they may also be produced by any other hetero-suggestion, *e. g.* the sight of a comrade with paralysis or anæsthesia, organic or otherwise. They may also be produced by auto-suggestion alone, and it is conceivable that this often plays a considerable rôle; from a tired feeling in a limb to its relative impotence there is but a step; thence to paralysis and anæsthesia there is but one step more, and these steps are small and soon taken, although in good faith, by neuropathic temperaments. The present exceptional circumstances do not appear to have altered these conceptions in any way; among the countless cases of purely functional disorder which I have observed since the beginning of the war, I have seen a large number in which there did not appear to be any question of simulation, but I did not find any case where the influence of auto- or hetero-suggestion could be eliminated."

Some persons have wrongly attributed to us the idea that we regarded these disorders as a fiction, or as the result of simulation. What we did say was very different, viz. that they were the result of suggestion.

It should also be added that some forms of organic hemianæsthesia may simulate those of hysterical origin.

Anæsthesia of a more or less segmental type may also be observed in Volkmann's syndrome, and in reflex paralysis and contractures (Gougerot and Charpentier (151), Sicard (157), Babinski and Froment (145)). They were formerly noted in cases of hæmorrhage into the spinal cord and syringomyelia, and have recently been found at the onset of hæmatomyelia following shell explosions (Froment) (205).

Fig. 1.—Neri's sign in a patient with left sciatica. When the trunk is bent forwards the knee is flexed on the affected side.

If anæsthesia corresponding to this type were always regarded as hysterical in nature, grave errors of diagnosis would be committed. Errors of this kind have undoubtedly led some neurologists to give hysterical anæsthesia the power to modify or abolish the cutaneous reflexes. We will discuss the cases brought forward in support of this view in a special section devoted to the question of the reflexes in hysteria (*v.* p. 471).

Hysterical pains.—Of all the hysterical pains, the most frequent undoubtedly is hysterical pseudo-sciatica, in which the signs of sciatica properly so called are absent, viz. loss of the tendo Achillis reflex, scoliosis, Lasègue's sign (pain caused by flexion of the thigh combined with extension of the leg), Neri's sign (*v.* Fig. 1), and Bonnet's sign (pain caused by adduction of the thigh).

Motor Disturbances

Hysterical paralysis.—Hysterical paralysis presents the most varied types, such as *hemiplegia, paraplegia* or *monoplegia* of the upper and lower extremities.

The most frequent of all these forms of paralysis appears to be paraplegia. It develops as a rule as the result of commotion due to a shell explosion accompanied sometimes by a fall or being buried in a trench. Although the above-mentioned causes can provoke hæmatomyelia (Jumentié (213), Ravaut (240), Heitz (211), Froment (205), Jean Lépine (221)), the communications of Pierre Marie and Mme. Athanassio-Bénisty (70), A. Léri (73), Roussy and Boisseau (109), in France, and of Bonhœffer and Karplus in Germany, as abstracted in the *Revue Neurologique*, have proved the purely neuropathic nature of a considerable number of paraplegias occurring under these conditions. Laurent had previously seen cases of the same kind during the Balkan War; and there is every reason for supposing that many of these cases, in which a cure was obtained rapidly, were due to hysteria. But hysteria is not always the only factor; the existence of sphincter troubles, of short duration, and certain changes in the cerebro-spinal fluid, indicate, even when counter-suggestion is decidedly successful, that there had originally been real organic disorders on which were grafted the pithiatic phenomena which persisted. Complete brachial or crural monoplegias are also observed, and not unfrequently motor disorders roughly simulating paralysis of the brachial plexus or musculo-spiral paralysis.

Typical astasia-abasia, simple or spastic, or other forms of systematic paralysis (*v.* p409) are not rare in the neurology of war.

We will not describe here the distinguishing features between these different varieties of hysterical paralysis and organic paralysis, as we shall deal with this question at length in the chapter on "Diagnosis."

Hysterical contractures.—Hysterical contractures are less frequent than was supposed during the first months of the war, when a large number of contractures were regarded as hysterical, though, as we have since shown, they should have been ranged among reflex nervous disorders. Genuine hysterical contractures,

however, which are cured rapidly and completely by counter-suggestion are seen after wounds in war. In the upper limb they appear in the form of claw-hand, ulnar pseudo-clawhand, or the so-called illogical clawhand; in the lower limb they are chiefly shown by extension of the leg on the thigh and by club-foot.

A. Léri and Roger (123) were of opinion that contractures in a position of extension were almost always of hysterical nature. But at the time of their communication little was known about reflex contractures, which, as we shall see, sometimes show a similar condition. As a matter of fact, the diagnosis of hysterical contracture is principally based on the absence of the objective signs peculiar to organic or reflex contractures, and which cannot be reproduced voluntarily. It was held formerly, it is true, that hysterical contractures and hypnotic contractures, which are both the same, were quite distinct from voluntary pseudo-contractures. It was claimed that if an attempt was made to correct the vicious attitude, there was no return to the old position or jerky movements which are always present in the latter. Some writers even have maintained that hysterical contractures may persist in natural sleep. But none of the cases which had been formerly brought forward in support of this view had presented all the features necessary to guarantee their authenticity.

What does the neurology of war teach us in this respect? All the cases of hysterical contracture which we had the opportunity of examining resembled voluntary contractures in every respect. If an attempt was made to overcome them, one noted the existence of a return to the old position, of jerky movements, of intermittencies and variations in their attitude, which were all the more frequent and pronounced as the patient's fatigue increased under the influence of the exercise. Moreover, if after carefully diverting the patient's attention, the contractured limb was suddenly seized, it was often possible without any

effort to overcome a contracture which had resisted the most vigorous traction a moment before.

Other observers have been struck by the same peculiarities. The following is the opinion of A. Souques (71): "Neuropathic contracture or pseudo-contracture is not accompanied by any disturbance of superficial or deep sensibility, of reflex action, or vaso-motor disorder. It cannot be distinguished by any clinical sign from voluntary contractions. Its localisation is roughly related to the position of the wound, but the vicious attitude usually bears no relation to physiology, but depends upon the personal ideas of the individual."

In a thesis inspired by Roussy Mme. Grutzhaendler-Judelson (77) wrote of hysterical contractures as follows: "All sorts of clawhands may be met with, including those which resemble that caused by a peripheral nerve lesion and others which are entirely illogical. One of their characters is to disappear in sleep, while an organic clawhand persists."

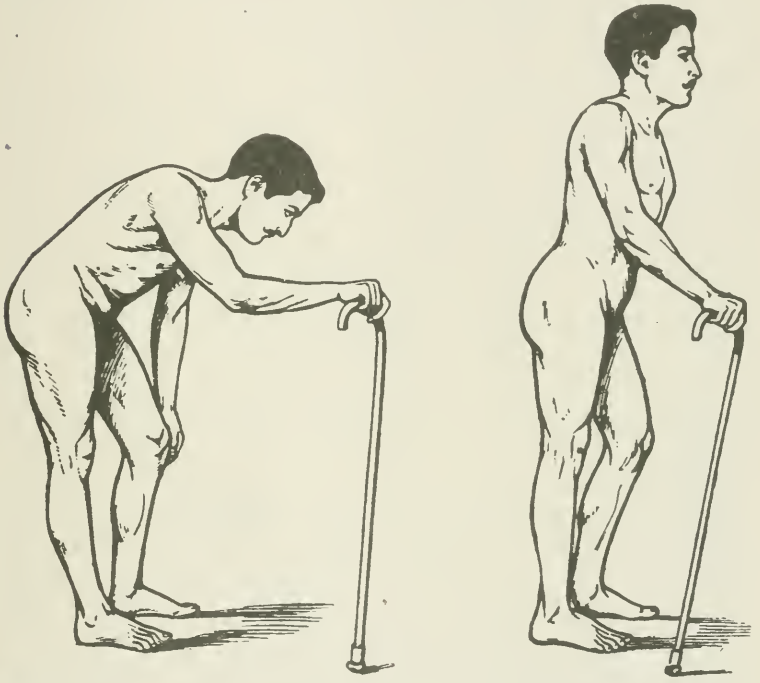
As to this last point agreement is not yet established. G. Ballet (84) has invented an arrangement the use of which he thought would enable him to settle the question in dispute. The following is the method which he adopted in a case in which he made the diagnosis of hysterical contracture: "I put a strip of stamp-paper along the inner side of the leg and foot. The perforations, separating the different sections of the strip, prevent it resisting even a slight traction. It was therefore obvious that if the foot, which was in a position of forced adduction, returned in the course of the night even in a very incomplete manner to a normal or nearly normal position, the strip would break. On the other hand, it is improbable that a patient who was not complaining of insomnia would remain awake the whole night, especially if, as was actually the case, he was encouraged to sleep by giving him six grains of veronal. It may, therefore, be granted that the unbroken condition of the strip in the morning is the proof of the persistence of the contracture during

sleep. Now, in the present case the band was not broken. The fact which I have just related, therefore, helps to show that some hysterical contractures at least persist during normal sleep." Other observations of the same kind have been reported by Sollier (89).

Are these facts really conclusive? As we have definitely stated before, it is essential to eliminate from the discussion all cases in which fibro-tendinous retractions are associated with the contracture. Examination under a general anæsthetic is the only certain means of recognising their absence. This method of investigation has also the advantage, as we have shown, of occasionally revealing certain peculiarities in the state of the tendon reflexes and muscular tonicity which help to exclude hysteria without any doubt (*v.* p. 503). In none of the above-mentioned cases was examination made during narcosis. The decisive proof, viz. disappearance by counter-suggestion, was also lacking. Lastly, we may remark that the individuals concerned had undergone traumatism; in G. Ballet's case, in particular, the contracture had developed after an operation on the first metacarpal bone. It is possible, therefore, that it was a case of a reflex contracture. Such contractures may show no change in natural sleep as Duvernay (124), Sicard (129), and ourselves have noted; they persist sometimes, as we have shown, until an advanced stage of chloroform narcosis; they are as persistent and fixed as the contractures associated with lesions of the nerve centres. Now, the diagnosis of reflex contracture had not even been considered in the writings with which we have just dealt. The facts in question, therefore, do not justify us in concluding that hysterical contracture properly so called behaves differently from a voluntary pseudo-contracture.

Vertebral curvature.—Following traumatism of the back and loins, as well as after commotion due to the explosion of large shells, one may observe curvature of the trunk (simple kyphosis or kypho-scoliosis),

which has been described by one of the following terms: "vertebral curvature," "vertebral antalgic attitudes (Sicard) (83), "traumatic kyphosis," "spondylitis," "contracture of the abdominal muscles," and "camptocormia" (Souques).



FIGS. 2 and 3.—Traumatic kypho-scoliosis following commotion by a shell explosion (August 1914), showing patient's attitude before (Fig. 2) and after (Fig. 3) the first treatment (July 1916). The improvement thus obtained progressively increased.

Souques and Mme. Rosanoff-Saloff (93) (100) have carefully studied its morphology. The modifications of the folds, dimples and muscular prominences in camptocormia, they say, are identical with those which accompany the physiological flexion of the trunk described by Richer. The only difference is that in the first case when the subject extends his head he shortens the nape of the neck, which presents several transverse folds at the same time that the vertebra

prominens disappears, whereas in the second case flexion of the head accompanies flexion of the trunk. But if a normal subject in this flexed position is told to extend his head, the same morphological changes in the trunk appear. Extension of the head, these authors add, appears to be necessitated by the need that the patient has of looking at a distance straight in front of him, so as to walk properly.

These contractures of the trunk are usually not accompanied by any objective sign of a lesion of the nervous system. It may be remarked, however, that they sometimes persist in the dorsal decubitus, and may even, exceptionally it is true, be accompanied by bony lesions which can be revealed by the X-rays. We may note incidentally that similar cases described in Germany by the name of severe hysterical kyphosis or scoliosis seem to be an exact counterpart of these cases. Photographs taken some time ago, which appear in Laurent's book on the Balkan War (217) illustrate the same type.

Without it being possible at present to describe exactly the pathogeny of these phenomena, we are convinced that there often existed originally certain lesions, usually of a mild character, which were the cause of the rigidity and peculiar attitude. This organic state which is often transitory has had grafted on to it hysterical disorders which keep up the deformity. As a rule, one succeeds in rapidly modifying the curvature under the combined action of psychotherapy and movement, but its complete disappearance is not always obtained at once. The kyphosis persists in a certain degree, and at first it has a fairly marked tendency to recur. Even when the patients are placed under the best conditions for treatment, complete cure often demands several weeks. At least this is the result of our own observations, as well as those of Clovis Vincent, in a large number of cases.

As regards the future of these patients and their ability to resist prolonged effort and fatigue we have not yet come to a decision, and to get accurate infor-

mation, we must wait for the observations made by regimental medical officers and by those belonging to units at the front. A general enquiry as to this particular point would doubtless furnish interesting results.

Claudication.—Hysterical claudication is not rare, and presents the most different types. First of all there is the claudication which it is impossible to describe, as it changes from one moment to another. It is sometimes enough for the person supporting the patient to pass suddenly from right to left or from left to right to make the limping undergo a complete change. The necessity of transferring the weight of the body to another side throws the patient off his guard, and he changes his claudication without realising it. The claudication often presents paradoxical forms, and in that case belongs more to the sphere of dancing than to neurology. For instance, a soldier wounded in the right lower limb bends his two knees at each step he takes, and thus reproduces the movements peculiar to certain Russian dances. Another imitates the pedalling of a bicyclist. Such cases merely constitute one of the varieties of rhythmic chorea (*v.* p 410).

Sometimes the hysterical character of the claudication is less obvious, and only appears on close examination. Such are the cases to which Meige (88) drew attention, in which the subject while walking retains an attitude of one suffering from hip disease, and presents an apparent shortening of the lower limb from inclination of the pelvis. This shortening disappears when the patient kneels or walks on his knees. Such again is another type of limping which resembles in some of its characters the gait of subjects with a "Spring knee." As a rule these show abnormal attitudes or movements caused in the first place by a transitory organic disorder and rendered permanent by habit. "Habit creates a tendency to habit," writes Meige. "A large number of anomalies in standing or walking," he adds, "are the result of bad habits caused by a pain or weakness, which though real is only

transient. During the time that the patient really feels this pain or weakness localised in the lower limbs or trunk, he instinctively adopts the position in standing or walking which is most likely to diminish the pain and the weakness. Consequently, when the pain or weakness has improved or even completely disappeared, the patient still retains his abnormal habit of standing or walking."

But it would be wrong to attribute to hysteria all the varieties of traumatic claudication which do not appear to be adequately explained by the lesion which has caused them. We have shown that a certain number of them belong to nervous disorders of a reflex character (*v.* p. 520). A minute examination is necessary in every case. By adopting this method in dealing with a patient presenting a claudication which did not correspond to any known type, we were able to discover by X-ray examination a fracture of the vertebral column in the lumbar region, which had escaped notice.

Tremors.—In the neurology of war obstinate tremors are very frequently observed, usually as the result of the shock caused by a shell explosion.

The sufferers do not present any sign of paralysis agitans, disseminated sclerosis, Graves' disease, cerebellar affection, general paralysis, mercurial or alcoholic intoxication, nor is there any question of hereditary tremor. These tremors are usually attributed to hysteria. H. Meige (98) protests against "the abuse made of the diagnosis of hysterical tremor to describe tremors of unknown nature." "The tremor observed after shock," he adds, "resembles those regarded as neuropathic manifestations and commonly seen in traumatic neurosis. It is not impossible, however, considering its etiology, tenacity and unchanging character that it is the consequence of a material change in the nervous system caused by a violent explosion."

G. Ballet (94) has noted that some of these tremors are accompanied by an expression of fear or alarm,

and appear to be associated with a mental disorder. Side by side with this peculiar mental state there is another, which would also help to make the tremor persistent, viz. the tremophobia described by Meige.

"The physical phenomenon," he writes, "produces the obsession, which in its turn amplifies the somatic reaction. The exaggeration of the latter reacts in like manner on the mental disorder; a vicious circle is thus formed of interchanging psycho-physical reactions, the final result of which is an obsession." In any case, we must recognise that we have here a problem which is not completely elucidated at the present time. Apart from those cases in which the tremor has been cured by counter-suggestion, and of which the hysterical nature was undoubted, there are others the mechanism of which is not settled.

The State of the Reflexes in Hysteria

No new case has been brought forward to throw doubt on the law which we primarily established to the effect that hysteria never modifies the state of the tendon reflexes and pupillary reflexes.

Is it the same with the cutaneous reflexes? None of the numerous cases of hysteria which we have observed has hitherto served to change our opinion. In a communication to the Neurological Society of Paris on February 4, 1915, Dejerine (75) brought forward three cases connected with the neurology of war in support of the contrary opinion. "The cases which I am bringing forward to-day," he said in conclusion, "appear to me to demonstrate absolutely that functional anæsthesia, whether due to hystero-traumatism or another cause, may in certain cases produce complete abolition or very great diminution of the plantar cutaneous reaction (plantar cutaneous reflex and movement of defence)." Other cases were brought forward in support of the same view by Paulian (72), Jeanselme and Huet (81), and Sollier (95). But

were these cases really conclusive? In the discussion following Dejerine's communication one of us made the following remarks :—

"If voluntary movements may be mistaken for reflex movements, on the other hand a reflex may be masked by a voluntary muscular contraction; this applies to cutaneous reflexes as much as to tendon reflexes. Many normal individuals by an effort of the will are able to keep the lower limb motionless when their sole is stimulated. Moreover, it is not uncommon for a subject who has reacted to the first or first few cutaneous stimuli to show no more reactions when the stimuli are renewed, and he is to a certain extent upon his guard. In examining these reflexes one should make sure that the leg, foot, and especially the toes do not become stiffened as the result of a voluntary contraction.

"Now, in Dr. Dejerine's first two cases this fundamental condition is not realised, far from it, since they both present contractures, on the intensity of which Dr. Dejerine actually insists. In the third patient, it is true, Dr. Dejerine shows that though the foot is in a state of contracture, the toes are not; but it was in this third patient, whom I have just examined, that I succeeded in obtaining a definite flexion of the little toes by stimulating the sole. In the two cases to which Dr. Dejerine alludes, and which are illustrated in his book (64), the plantar cutaneous reflex appeared to be abolished, and the muscles were also in a state of contracture. To sum up, out of the five cases related by Dr. Dejerine in which he thought the reflex was abolished there were four with contracture; in the fifth, in which contracture of the toes was absent and was nevertheless accompanied by hysterical anæsthesia, the reflex movement could be produced. These cases therefore cannot serve to prove that hysterical anæsthesia is capable of abolishing the plantar cutaneous reflex. They serve rather to establish the contrary, and to show that there is only an apparent absence due to hysterical contracture which is as capable as

voluntary muscular contraction of masking reflex movements."

To this criticism we may add another which is of fundamental importance In almost all the cases mentioned above, as far as one can judge by the brief account which is sometimes given, the patients had undergone various forms of traumatism, such as wounds of the limbs by a projectile, contusion, or commotion by shell explosion. It is very possible, therefore, that the nervous troubles incriminated were not hysterical but reflex in nature. As cases of this kind had not attracted attention at the time of Dejerine's communication, this diagnosis was not even discussed. Now, in the so-called reflex pareses and contractures, as we have often noted, the plantar cutaneous reflex may be abolished, the loss of the reflex being apparently connected with vaso-motor troubles and hypothermia. It is enough to warm the foot artificially and sometimes only to leave the patient in a warm room to see the reflex return. It is important to emphasise the instability of this reflex, and we seize this opportunity to repeat once again that the rapid disappearance of a phenomenon by no means implies the idea of hysteria, as some suppose. It only has this signification when the disorder has yielded to the sole influence of counter-suggestion (*v.* p. 559). To sum up, the new cases which have been brought forward to prove that hysteria is able to abolish the cutaneous reflexes are anything but conclusive.

CHAPTER IV

SYMPTOMATOLOGY (*continued*)

Disorders of Special Sense

Hysterical visual disorders.—We asked Dr. Morax to tell us about his observations on hysterical visual disorders since the beginning of the war, and to give us his present opinion as to the "ocular stigmata" of hysteria. We applied to him because in addition to being an ophthalmologist he possessed a special knowledge of nervous diseases, having formerly been attached to Charcot's service at La Salpêtrière, and was therefore particularly qualified to give an opinion on the subject in question. The following is the note which he kindly sent us—

"The visual troubles formerly attributed to hysteria may be divided into two groups, one comprising the phenomena which are present before any medical examination, like blepharospasm, amaurosis, etc., and the other the symptoms unknown to the individual and which a methodical examination alone brings to light, or seems to bring to light, like narrowing of the visual field, monocular polyopia, dyschromatopsia, etc., which were regarded as stigmata of hysteria.

"It appears to me advisable to make this distinction at the beginning of this note, and to state that, while the manifestations of the first group retain all their nosological importance, it is not the same with the others. Their reality has been rightly disputed, and what we have seen during these two years of war is in agreement with this opinion. I will also draw attention to the extreme rarity of hysterical ocular manifestations, in spite of the frequency of cephalic and general

traumatism, and in spite of the introduction of new methods, such as the use of lacrymatory gas, which is liable to act on the imagination and to cause visual suggestion. Among many thousands suffering from diseases or wounds of the eyes I can find only a few cases which can be attributed to hysteria.

"*Ocular hyperæsthesia and blepharospasm.*—Among other disorders lacrymatory gas causes a change in the corneal epithelium which produces the hyperæsthesia and pain like that resulting from a foreign body imprisoned between the lid and the eyeball. The sensitiveness to light is extreme, resulting in a very marked reflex lacrymation and a contracture of the orbicularis palpebrarum. Under normal conditions the repair of the epithelium takes place rapidly; usually in less than a week, rarely in more than a fortnight, everything is all right again.

"I had the opportunity of observing, under conditions which I am going to describe, a wounded man in whom the phenomena of ocular hyperæsthesia, photophobia and blepharospasm persisted in an abnormally prolonged manner. As he had been nursed in an auxiliary hospital for several weeks with his head and eyes covered with a voluminous dressing, and was the object of the sympathy of the female staff, who shared his fears that he would remain blind, he was in the most favourable circumstances for submitting to the suggestion of his entourage. Although he presented no ocular lesion, the removal of the bandage was followed by so much lacrymation and photophobia that a new one was at once applied. Examination of the eye and the visual function after superficial anæsthesia by cocaine drops did not show any pathological change.

"I drew attention to the harm and danger of an eye-dressing in this case, reassured the wounded man by telling him that the eye lesions produced by gas were not serious, and in a few days the trouble had disappeared.

"*Hysterical amaurosis.*—In hysterical amaurosis the peripheral visual apparatus and the pupillary

light reflexes are perfectly normal. The affection is only revealed by what the patient says and by the fact that he behaves like a blind man.

"After a superficial wound of the scalp a patient showed complete blindness lasting for four months until the surgeon performed craniectomy. The following day vision began to return and continued to do so until its restoration was complete, without the appearance of hemianopsia, lesions of the fundus, or dyschromatopsia. I did not see him when he was blind, and examined him only after he had recovered; but I cannot explain the case except by supposing that the amaurosis was hysterical; the wound of the scalp had been quite superficial and had healed in a few days. The absence of any fracture or fissure of the bone was shown at the craniectomy. When a meningeal or cerebral lesion causes blindness it may be due to œdema of the optic nerve and hypertension of the cerebro-spinal fluid. Œdema of the optic nerve which has lasted four months always leaves changes in or near the discs visible on ophthalmoscopic examination. In this case the absence of such changes allowed me to conclude that the amaurosis was of an hysterical nature. When I examined the patient the retinal sensibility, the visual fields and colour sense were normal.

"*Spasm of accommodation. Spasmodic myopia.*—There has been described under the name of spasm of accommodation or spasmodic myopia a special state of the vision which appears peculiar to hysteria, for it is not observed in any organic affection of the eye. In this condition the refraction of the eye undergoes an increase similar to that which is produced by the refractive change of the lens in the act of accommodation. The patient, whose distant vision was previously normal, no longer clearly distinguishes distant objects unless a concave glass is placed in front of his eye. His eyes have become myopic; but it is a peculiar myopia, paralysing the accommodation by atropine being sufficient to make the refraction normal again.

"As the result of a shell explosion at a certain distance from where he was, and without having been hit by the fragments, a soldier lost consciousness and was taken to a hospital in the south. According to what he told me, for I did not examine him at this period, he was deaf and blind. After several months the deafness disappeared and the vision partly returned, but he still had much difficulty with distant objects, although reading had become possible again. A month previously he had been examined by a very competent medical man, who had found a myopia of -7 D, and had ordered him the glasses which he had since been wearing. With these glasses distant vision was only $\frac{1}{3}$–$\frac{1}{2}$, and he was sent to us for further examination. I found that the glasses which he was wearing gave him the best distant vision, but I was puzzled by his blindness and deafness, and decided to make a complete examination of the refraction under atropine. The new examination showed me that the refraction of the eye was normal and the visual acuity = 1 without glasses. I may add that apart from the spasmodic myopia the visual function was perfectly normal.

"I have related these cases to show that the hysterical visual troubles observed in this war do not show anything to differentiate them from what we find in civil practice.

"*The so-called ocular stigmata of hysteria.*—As I said before these are signs which have to be looked for, as the subject does not present them unless he is examined in a special manner.

"*Concentric narrowing of the visual field* was rarely absent when it was given a special significance. To demonstrate it, a perimeter was used, care being taken to cover the eye which was not examined. Examination of the visual field under these conditions is an infinitely more delicate matter than it may appear. The causes of error are numerous, and among the most important must be mentioned the difficulty of the patient and observer understanding each other without the former submitting to the latter's sugges-

tion. If care be taken to control the result of a perimetric examination by methods which are apparently less precise, but are really much more accurate, such as the study of the visual field, not with an index but with familiar objects, the finger, etc., it will be found that the results of these two examinations do not agree. Formerly I had many cases of narrowing of the visual field in hysterical patients at La Salpêtrière, but now that I have had more experience in this examination, I do not hesitate to say that none of my previous findings appears to me to-day to be able to bear criticism, and I have not found any concentric narrowing of the visual field from retinal anæsthesia of neuropathic origin for a very long time, either in civil practice or in military practice during the last few years.

"I must say the same of the peculiar *dyschromatopsia* attributed to hysteria. In examining the visual field with a coloured index, the field of perception of red is found to be more extensive than that of the blue. If examination of the visual field with a white index is difficult, examination with a coloured index is so difficult that few oculists have been successful with it, owing to its being almost impossible to make an examination with indexes of the same distinctness. To demonstrate *monocular polyopia* a match was brought close to and moved away from the eye. Under ordinary conditions, if accommodation is normal and the object removed is in the field of accommodation, it appears single. If, on the contrary, the change in accommodation does not correspond to the movements of the object, it appears double. As I have said before, there is sometimes a state of spasm of accommodation which may explain this phenomenon, but this is a rare disorder, and in looking for it care must be taken not to suggest the reply by the manner of putting the question.

"To summarise my present views as to the nature of the visual disorders described as stigmata of hysteria, I will say that the doctor who avoids suggestion in

questioning his patients will have no chance of finding them."

It is obvious that the conclusions of Morax are identical with those drawn up by one of us (*v.* p.425). We will remind the reader that concentric narrowing of the visual field was formerly considered as a cardinal symptom, and occupied the highest rank in the hierarchy of the signs of hysteria. To show the importance attached to it by the partisans of the traditional doctrine, we will quote some passages from a recent book by P. Janet on *The Mental State of Hystericals.*

" I will emphasise only one special point which has always keenly interested me—namely, contraction of the visual field. The visual field is a whole or a system of sensations perceived simultaneously which contracts in the same manner as the field of consciousness. The sensation which serves as a centre round which all the others are grouped, the one which always enters most into consciousness, is naturally, save in exceptional cases, the clearest visual sensation, or the one which corresponds to the point of fixation. . . . Although the visual field is concentric as a rule, it is extremely variable in its extent; as it widens out it seems to follow the modifications of the patient's mind; in some cases it is a sort of barometer for hysteria.

" In short, in a very large number of cases in weak-minded persons, the effort of visual attention very considerably narrows their visual field.

" This finding is not without interest; from the clinical point of view it furnishes an element of diagnosis and enables one to find this important hysterical symptom, narrowing of the visual field, when it is hidden.

" Narrowing of the visual field may be regarded as the emblem of hysterical sensibility in general " (54).

When the former doctrine of hysteria is compared with the conclusions resulting from our own observations, which are confirmed to-day by the neurology of war, one can realise the distance separating the modern from the old conception of hysteria.

Disorders of hearing.—Hysterical deafness is one of the most frequent phenomena in the neurology of war. It is generally bilateral and apparently complete; it develops after commotions due to the explosions of large shells. When left to itself, it may last very long without undergoing any change: cases of deafness which have lasted eight to ten months are not rare. The persistency of deafness due to a psychical cause is all the greater, as it is often not recognised, and patients suffering from it are considered and treated as if they were really deaf. One frequently finds noted on their case sheet, "incurable deafness," or "permanent deafness." How is the chronicity of such disorders to be wondered at when medical suggestion thus adds its weight to those of auto-suggestion and corroborates it? And yet, however old the trouble may be, as soon as it is recognised and treated in the proper manner, it gives way as a rule rapidly and completely, sometimes even in a few moments.

This view is confirmed by Lannois and Chavanne, who cured fifteen out of thirty-two cases of deafness in one sitting. Contrary to what might be supposed, it is occasionally difficult to distinguish hysterical deafness from some forms of organic deafness. Organic war deafness (Lermoyez (223), Lannois and Chavanne (215)) may be due to various lesions; they consist of direct traumatic deafness, following fracture of the skull and the petrous portion of the temporal bone, and indirect traumatic deafness due to labyrinthine commotion, either pure or associated with other lesions of the ear (purulent otitis media, rupture of the drum). Lesions of the middle ear are easy to recognise, but it is not the same with pure labyrinthine lesions, which may be difficult to detect.

There is often some difficulty in establishing the diagnosis, and even very competent aurists may be in doubt. Patients of this kind have several times been sent to us with the request to examine them for the stigmata of hysteria to decide this question. Now, as we may remind the reader, such an examination

would not help to settle this problem. Moreover, it was fortunate that we did not pay any attention to the stigmata, for they were often absent in cases of hysterical deafness, and sometimes present in subjects suffering from labyrinthine commotion.

On what symptoms, then, can we depend for establishing a diagnosis?

E. J. Moure and P. Pietri, in an interesting study (235), have given a series of tests to which aurists should have recourse in such cases. We will confine ourselves to mentioning some of the methods which may serve as a guide to medical men who are not specialists.

It should be stated in the first place that a change in the voice, such as a trumpeting or uncontrolled voice presenting sudden and inopportune changes in tone and intensity, may be sufficient to characterise true deafness. But this peculiarity is far from constant; it appears to be frequently absent in war deafness, though the reason for this is not properly understood. The absence of this sign, therefore, does not allow us to dismiss the hypothesis of organic deafness. Other symptoms justify more reliable conclusions.

When a noise occurs suddenly, it is not uncommon to detect in a subject of psychical deafness an involuntary movement which is often quickly repressed and shows that the deafness is not real. Gault (208) has recently shown the practical value of the cochleo-orbicular reflex; a moderate acoustic stimulus (a bicycle horn blown six feet off) causes in hysterical deafness, as in the normal condition, a contraction of the orbicularis palpebrarum which is entirely absent in complete organic deafness.

There is another diagnostic method suitable for deaf persons who have been taught lip-reading and which enables one to detect "sham reading." To succeed in this it is sometimes sufficient to mask the movements of the mouth, in an apparently inadvertent way, while speaking. If this act is cleverly carried out, and if the subject of psychical deafness is not too

much on his guard, he will continue to follow the conversation as if nothing had happened to prevent him. The wounded may also be subjected to the very ingenious and simple test invented by Mr. Thollon, Inspector of the National Deaf and Dumb Institution (243), in which the patient has to repeat syllables or words pronounced in front of him and selected from those which the deaf lip-reader often confounds with one another.

"From a superficial point of view," writes Mr. Thollon, "it may be said that each element of speech has corresponding to it a mouth pattern, knowledge of which supplies a speaking deaf-mute with the key to lip-reading, just as knowledge of letters of the alphabet supplies the key to print or manuscript. But more careful consideration shows that things are not so simple. . . . Each sound is dependent at once on the action of the vocal chords, the tongue, lips, and soft palate, and relies at once on sight and touch. This description establishes such definite distinctions between the various phonetic elements that no confusion is possible. But when it is reduced to the features furnished by the play of the lips, tongue (often invisible), lower jaw and physiognomy, it is inadequate, and confusions become not only possible but easy and inevitable. Physical lip-reading is insufficient to allow a deaf man to repeat a phrase pronounced before him, psychical reading must intervene and the general sense of the phrase must allow him to guess the sounds and syllables which he has been unable to distinguish. By lip-reading alone it is impossible to distinguish from one another the consonants belonging to the group *p*, *b*, *m*. It is the same with the following groups of sounds: *t*, *d* and *n*; *e*, *g*, *gn*, *r* (guttural) and *y*; *f* and *v*; *s* and *z*; *ch* and *j*; *l* and *r* (lingual); *a* and *ar*; *eu*, *un*, *o* and *on*; *ou* and *v*; *i* and *é*."

"The same mouth image," continues Mr. Thollon, "may express, for example, *chapeau*, *chameau*, *jabot*, *Japon*, *jambon*, or *palais*, *ballet* or *malin*."

The person who is really deaf, not being able to distinguish the different words by lip-reading alone, will inevitably make mistakes, if he is not guided by the words forming part of a phrase.

This peculiarity will enable one to detect simulated or hysterical deafness. When the subject is made to repeat a series of isolated syllables or words containing those sounds which cannot be differentiated by sight only, it may be affirmed that, if he makes no mistakes, it is not a case of complete organic deafness.

In addition to the previous characters by which hysterical deafness can sometimes be differentiated from organic deafness, there are others which may supply the means of resolving the problem; we mean the signs indicating a perturbation of the vestibular apparatus. The existence of such a disorder, though it does not give us the right to declare that the deafness is also organic in nature, makes this hypothesis at least probable. A completely normal condition of the vestibular apparatus, on the contrary, leads us to suppose that the deafness is psychical.

For some years the diagnosis of disorders of the vestibular apparatus has acquired a certainty and precision which were lacking when it simply rested on the interpretation of subjective troubles. To-day it chiefly depends on the changes which occur, as the result of pathological conditions, in objective reactions to various stimuli, as in the gyratory (Mach, Egger), caloric (Barany) and voltaic tests (Babinski) (61). To these signs may be added the loss of orientation and equilibrium caused by various walking exercises (Stein, Babinski and Weill, E. J. Moure (234), Cestan, Descomps and Sauvage (191)).

We must emphasise the value of the voltaic vertigo test which appears to be less well known by aurists than other methods of examination. In order to investigate voltaic vertigo, one proceeds in the following manner: the electrodes of a voltaic apparatus are placed on the temples or mastoid processes, one on each side, and an electric current of a few milliamperes

is passed. This causes *in the normal condition*, among other phenomena, a feeling of giddiness, nausea, nystagmus and a lateral inclination of the head to the side of the positive pole. A rotation of the head is produced, and sometimes also rotation of the trunk by placing the electrodes as follows : the one connected with the negative pole behind the lower jaw, and the other above the tragus. The head executes a movement of rotation from the side of the positive pole beginning at the time of the closure of the current and continuing slowly. This movement is a physiological phenomenon, but is not constant ; it is more or less pronounced according to the individual, sometimes it occurs by itself and sometimes it is associated with inclination of the head. The following are the various modifications of voltaic vertigo which are observed in labyrinthine disorders and do not occur in hysteria : increase of resistance to the stimulus caused by the voltaic current, movement of the head backwards and forwards, replacing inclination or rotation ; cephalic nystagmus ; unilateral inclination or rotation ; inclination and rotation occurring on one side only ; unilateral inclination to one side with unilateral rotation to the other, and occasionally circumduction of the head. The value of these signs has previously been confirmed by several observers, and quite recently Moutier (236) has emphasised their importance. The following is a quotation from his work on the subject.

" The tests generally employed have been Babinski's test or voltaic vertigo and Barany's test or caloric vertigo. I do not hesitate to give the preference to the former. Its technique is extremely simple ; its various stages are readily controlled, and the results are particularly easy to compare. . . .

" *Systematic exploration of the labyrinth*, apart from its scientific importance, also furnishes conclusions of immediate interest. It proves, in fact, the sincerity of numerous wounded men whose subjective troubles would otherwise appear doubtful, and thereby facilitates the preparation of the necessary documents for pro-

posing discharge from the army or transfer to the reserve."

The results obtained by psychotherapy have seemed to us to agree with the conclusions furnished by the test of voltaic vertigo. If the vertigo was normal the deafness yielded in most cases to counter-suggestion, whereas this has only been partially successful (hystero-organic association) or a complete failure when the characters of the voltaic vertigo were altered.

Mutism.—Hysterical mutism occurs either alone or in association with deafness. It is just as curable as psychical deafness, if not more so. It may disappear spontaneously; sometimes the dumb man in the course of a dream begins to speak or cry, he hears his own voice and as soon as he wakes declares that he is cured. But, as a rule, the mutism persists unmodified for months, if it is treated improperly. Various treatments have been proposed which are all equally efficacious and whose mode of action is always due to counter-suggestion, whether this be applied voluntarily or involuntarily. Contrary to what is the case in deafness, hysterical mutism is easy to recognise. Charcot has given a masterly description of it (*v.* p 410). Exceptionally it has been possible to mistake a case of aphasia whose language is extremely reduced for mutism, but the opposite error is more frequently committed; it is, however, fairly easy to avoid. Unlike the mute who emits no sound the motor aphasic can always articulate a few words or syllables; he may even emit under certain conditions all or almost all the sounds which constitute oral language (Froment and O. Monod (200)). Written language is always intact in the mute, while it is more or less affected in the motor aphasic, without even excepting the pure motor aphasic (Froment and Mazel (199), Froment and Pillon (201), Pélissier (238)). Finally, and this last peculiarity should not be neglected, because it may be a guide to diagnosis from the beginning of the examination, the faculty of pantomime is remarkably intact in the mute, while on the contrary in the motor

aphasic it is poor, monotonous and unable to supply the defects of language. It may almost be said that a person suffering from defects of language who succeeds in making others understand, by a series of varied and expressive gestures, the various circumstances which accompanied his wound is not an aphasic but a mute. As regards hysterical aphasia, no case has been published to our knowledge since the beginning of the war. A certain number of cases which had been recorded previously should be accepted with caution.

Œdema, Vaso-motor and Trophic Disorders attributed to Hysteria

No neurologist was able to bring forward a single well-established case of *hysterical œdema* at the time of the discussion in 1908. The old conception seemed to have arisen from an error of diagnosis, simulated œdema artificially produced by a voluntary constriction and œdema associated with organic affections having been attributed to hysteria.

Since the beginning of the war we have not observed any case of œdema which could properly be attributed to hysteria. A certain number of traumatic œdemas which would doubtless have been considered formerly as hysterical were due to voluntary constriction of a surgical nature, as in the cases described by A. Léri and Roger (85). Sicard and Canteloube (103) made a further study of these " constriction œdemas " recently and distinguished two stages in their course; in the first stage the œdema disappears as soon as the constriction ceases, but in the second period it becomes organised and its disappearance is then slow; it may even become permanent.

Some cases, however, of hysterical œdema have been published by G. Ballet (82), Lebar (86) and Raynaud (87). One of them provoked a discussion in the course of which Roger made the following remarks: " The question of hysterical œdema seems to me to be

settled for the present. Since Babinski's work everybody is agreed that this form of œdema does not exist. Cases of the kind always indicate trickery on the part of the patients or an organic lesion of vascular or nervous nature. In Dr. Raynaud's patient, who presented a wound on the inner aspect of the upper arm, just at the level of the brachial nerves and vessels, the existence of an organic vascular lesion seems at least probable and would explain the œdema observed."

To these hypotheses may be added another, viz. that of reflex vaso-motor disorders. In three out of four of the cases published, the œdema was associated with a contracture and had developed in an injured limb. Attention has already been drawn to this condition in dealing with the question of persistence of contractures during sleep (*v.* p 466). We may add that in none of these cases did the therapeutic test give a positive result; in Lebar's case the œdema and contracture diminished after superficial scarifications, but did not disappear entirely. This mode of treatment, it is hardly necessary to state, cannot be regarded as having an exclusively psychical influence.

Vaso-motor disorders were formerly considered very common in hysteria. We contested this opinion, and a large number of neurologists adopted our point of view, although a final agreement was never reached. It may have been thought at first that the cases observed at the beginning of the war were in favour of the old doctrine, as many patients with symptoms which were regarded as hystero-traumatic presented marked vaso-motor disorders. But further examination led to their being separated from the hysterical group, and to their being regarded as reflex phenomena due to disturbance of the sympathetic system (*v.* p. 528). This question, which is of the utmost importance both from a practical and a doctrinal point of view, will be discussed later in detail. We will content ourselves by saying, what we hope to establish later on, that though an hysterical paralysis may cause a slight

thermo-asymmetry it never produces definite vaso-motor or thermic disorders.

In spite of the frequency of hysterical phenomena not a single case has been seen of erythema, vesiculation, ulceration, gangrene of the skin, hæmorrhage, hæmatemesis, anuria, or any of those symptoms the reality of which was formerly admitted without discussion.

Sollier (96) noted, it is true, the existence of trophic bone-changes in six cases of so-called neuropathic contractures which developed after wounds. But such phenomena have often been seen after fractures and injuries of the limbs in the absence of any hysterical manifestations. It may be questioned whether these were not trophic changes of a reflex character associated with disorders of the sympathetic system.

Apart from these observations, which we hesitate to accept, *no case of hysterical trophic changes* has been published since the beginning of the war. In this connection we may note that according to Cestan (192) changes in the finger prints are exceptional in functional paralyses. "They are never found," he adds, "except in cases where the neurotic phenomena have been produced by an organic factor.". Villaret (245) also remarked that the trophic troubles of the skin, hair, nails, etc. (hyper- or hypo-trichosis), are absent in so-called functional paralyses, with the exception of cases corresponding to those which we have classified under the name of reflex paresis and contracture.

We think we are justified in concluding, therefore, that the modern conception of hysteria has not been shaken in any particular. On the contrary, it has been confirmed by numerous observations made during the last two years.

Although the contrary opinion has been held by some neurologists, this is doubtless due to the fact that the injuries of warfare have given rise to a number of nervous accidents which were at first misinterpreted and attributed, in the absence of a precise semeiology, to hysteria, although they were absolutely distinct

from it. In this respect there has been a confusion of ideas similar to that which existed in regard to symptoms connected with lesions of the central nervous system before their characteristics had been clearly determined.

The following chapters will be devoted to a study of these reflex phenomena, concerning which we have learned much that is new and for which a special place must be reserved in nosology.

PART II

REFLEX NERVOUS DISORDERS

CHAPTER V

HISTORICAL

Among the nervous phenomena observed in the neurology of war we must isolate a group of fairly numerous cases which present special clinical characters and which possess a definite autonomy. They include contractures, paralyses or paretic states which develop after traumatism. The motor disorders are not accompanied by any of the signs which are characteristic of typical organic affections, such as lesions of the central or peripheral nervous system, or lesions of the large vessels. They resemble in some features hysterical manifestations, inasmuch as the lesion which has caused them appears sometimes very slight and out of proportion to the resulting disturbance of function; they tend to spread beyond the limits of any known anatomical area; they show little response to treatment, and, unlike pithiatic phenomena, resist the influence of counter-suggestion.

They are distinguished from hysterical manifestations by various symptoms, all of which may not be present at the same time. In addition to contracture and paralysis, which may be associated though usually situated in different segments of the limb, the complete syndrome presents: muscular atrophy, exaggeration of the tendon jerks, changes in the cutaneous reflexes, which may amount to actual loss, hypotonus, mechanical hyperexcitability of the muscles with slowness of

the muscular contraction, quantitative changes in the electrical excitability of the muscles (increase or diminution in excitability without R.D.), mechanical and occasionally electrical increase in excitability of the nerves; disturbance of objective and subjective sensibility (anæsthesia and pain); disturbance of thermotaxis (especially hypothermia) and vaso-motor control (cyanosis, salmon-pink tint, diminution in the amplitude of the oscillations at the periphery of a limb when the atmospheric temperature is low); secretory disorders, and, lastly, trophic disorders of the bones, skin, hair and nails.

These various symptoms form combinations which differ according to the number, mode of association, and actual importance of each of the constituent elements. They give rise to various clinical types which, in spite of their differences, apparently belong to the same family and represent a nosological species.

We will set forth later the reasons which seem to us to justify these phenomena being considered as reflex in nature, and we will state what should be understood by this expression. But while admitting that the pathogenesis is still unsettled, the disorders in question, whatever names they may receive, constitute a special group, halfway, as it were, between organic affections properly so called and hysterical disorders.

These phenomena may be entitled *physiopathic*—a term intended to express the idea that, on the one hand, neither hysteria nor any other psychopathic state can produce them, and, on the other, that while indicating a physical and material disorder of the nervous system they do not appear generally to correspond to any nervous lesion which can be detected by the methods at our disposal.

Historical

Old views.—How was this nosological group formed? It originated a long time ago, and dates back to the work of Hunter, Charcot and Vulpian.

John Hunter (111) was the first to call attention to weakness and atrophy of the muscles which occur as the result of lesions of the joints. "It is remarkable," he states, "that an injury done to the tendons, ligaments, fasciæ, etc., especially of the strain kind, impairs the muscles more than when the muscles themselves are injured."

These early observations were confirmed and completed by a series of studies. We will merely mention the paper in which Gosselin (112) described muscular atrophy following fracture of the long bones, Ollivier's thesis (113) on muscular atrophy, the communications of Le Fort to the Surgical Society in 1872 and 1876, with the interesting discussion which followed, and Valtat's inaugural thesis (115), which consists of a clinical and experimental study and a detailed historical account. Lastly, a remarkable description of reflex disorders is to be found in the lectures of Vulpian and Charcot.

Charcot (119), in a study of the atrophies following lesions of the joints, showed that atrophied muscles presented a mere diminution of electrical excitability, or, in other terms, purely quantitative and not qualitative changes in the electrical reactions (such were also the results of Erb and Rumpf's researches). This amyotrophy is thus distinguished from degenerative atrophy. It is usually localised in the extensors, and is accompanied by a paretic state of varying intensity. The motor disorders are not always so closely circumscribed, and paresis of the foot sometimes occurs after traumatism to the knee. In such cases contractures may be observed with or without amyotrophy or amyotrophic paralysis.

Exaggeration of the reflexes appears to be a symptom common to the various forms of motor inhibition, which, as Charcot adds, are liable to develop after any joint affection, whatever its nature, intensity, and mode of evolution. "There is no necessary relation between the intensity of the joint affections and that of the paralysis and atrophy. . . . The persistence of

the deuteropathic symptoms (paralysis and atrophy) after the cessation of the protopathic affection (arthritis) is more or less the rule. . . . Months may elapse with the limb still useless, whereas the arthritis has for a long time only been manifested by a slight thickening of the periarticular tissues, if, indeed, there be even as much as that left."

Vulpian supplements this description as follows: "The sensibility of the skin and deep parts may be modified. Dr. Descosse in cases of arthritis of the knee has found evidence of neuralgia of the corresponding anterior crural nerve. The nerve was the seat of spontaneous pain, and pressure along its course produced a sort of numbness rather than pain in the corresponding area."

Vulpian emphasised the trophic disturbances of the skin, hair and nails, the secretory disorders, the coldness of the limb, and the cyanotic or dull pink colour of the skin. He pointed out that wounds accidentally inflicted on a limb affected by muscular atrophy are extremely slow in cicatrising, and even present a tendency to ulceration. Lastly, he adds that all peripheral lesions, including frost-bite, burns, and more or less deep wounds of the limbs, may become the starting-point of such phenomena. The following is a remarkable description of nervous disorders of this kind following wounds by firearms, resembling some of the cases which we had the opportunity of observing in this war. We think, therefore, that the passage should be quoted in full.

"Muscular atrophy is often not the immediate result of the peripheral lesion which gives rise to it. A gunshot wound, for example, in which only the soft parts of the leg have been involved, may cicatrise completely without the muscles of the leg showing any appreciable atrophy. Some months or one or two years after recovery the scar becomes painful as the result of a shock or fatigue or without any known cause; the pain becomes more or less intense and continuous, and is often accompanied by attacks of exacerbation. The pain seems to start from the old wound and to pass up the limb along the course of the

nerves.[1] Then the patient notices after one to three months that his leg is diminishing in size, and that it is the same with his thigh and buttocks. At the same time he has increasing difficulty in walking; he can at first just raise his toes from the ground, but later he is quite unable to do so; he drags his foot along the ground or walks on tip-toe, and is obliged to use a stick. If he has made careful observations on himself, he calls the doctor's attention to the changes in the coloration of the skin and shows that his foot is quite cyanosed when he is standing. Coldness of the leg and changes in the hair of the limb are observed; the skin is fairly often moist, especially in the lower part of the affected limb, while it may be dry on the sound side. When the lower limb is affected a more or less marked œdema of the lower part of the leg may sometimes be seen after a walk of any distance. These changes take from four to six months to develop, and continue to occur after the scar has become painless again, and all the pain has ceased in the limb.

"If the state of the muscular system be examined, it will generally be found that there are no fibrillary or fascicular contractions. Idio-muscular contractility

[1] Vulpian refers, in this connection, to Case CXLV (p. 745) in his Clinical Lectures at the Charité, where we find the following description: A soldier had been wounded on the outer aspect of the right leg at the junction of the lower and middle third, on August 6, 1870, at the battle of Reichshoffen. He was taken to a hospital at Chalôns, from which he was discharged at the end of August. He was able to resume his duties and take part in the Sedan campaign as well as in the military operations of the siege of Paris. At this period the wound, which appeared to be cicatrised and seemed only superficial, began to show some slight suppuration again. When he left the service in 1873 he joined a firm as accountant. In June 1875 he suffered from pain and a feeling of numbness in the wounded limb. The symptoms soon became complicated by a pronounced motor weakness, for which he was admitted to hospital in June 1876. Vulpian then found an amyotrophy which was most marked in the thigh, without obvious change in the electrical reactions, and pronounced vaso-motor phenomena. These nervous symptoms rapidly yielded to treatment, and six months after admission to hospital there was decided improvement.

may be increased. Electricity has less action on the muscles of the buttocks, thigh and leg on the affected side than on those of the sound side.

"Muscular atrophy in cases of this kind is very difficult to cure, and, as a rule, only very incomplete results are obtained."

We find very definite references in these descriptions to several of the principal features of the clinical syndromes which we are studying, viz. muscular atrophy with absence of R.D., paresis and contracture, either alone or combined, changes in the tendon reflexes, sensory disturbances (anæsthesia, neuralgia and pain on pressure on the nerve trunks), vaso-motor thermal and trophic disorders, and disturbance of the secretion of sweat. Moreover, it is noted that these phenomena may have an articular or abarticular origin, to use Dutil's expression; that they often seem out of proportion to the lesion which gave rise to them; that they sometimes develop late after complete cicatrisation, and, lastly, that they are usually very unresponsive to treatment.

For the sake of clearness, and in order to limit the subject which we have to discuss, we must anticipate a little of what we have to say about the pathogeny, and remind the reader of the meaning which Charcot and Vulpian gave to the term "reflex," when they applied it to the disorders in question. "The favourite theory," said Charcot, "with most contemporary writers appears to be this: the articular affections reflect certain irritant impulses along the articular nerves to the spinal cord, which impulses modify the trophic centres in that organ, whence emanate the motor nerves and the nerves which regulate the nutrition of the muscles." These ideas were admitted without discussion at that time by the majority of neurologists. No critical work to our knowledge has appeared since then to throw any doubt on the above descriptions.

Nevertheless, to judge by the place given to this subject in most modern text-books of neurology, it does not seem that the same importance has been

attached to it lately, and it may even be said that a certain amount of discredit has fallen on cases of this kind.

To what must we attribute this evolution of medical doctrine? It appears to us to be due to the following two causes: first, to the abuse which some writers have made of the term "reflex," and, secondly, to the extraordinary vogue of the idea of hysteria. We shall now try to illustrate these two points, before engaging in the study of cases recently observed in the neurology of war.

Formerly the name of "sympathetic paralysis" was used to describe a somewhat heterogeneous group of paralyses caused in certain regions of the body by affection of a more or less distant organ. These were the paralyses which Graves and Brown-Séquard afterwards called "reflex paralysis," and which Jaccoud and Weir Mitchell proposed to call "paralysis of peripheral origin," or "paralysis from peripheral irradiation." It is most important to realise that these are cases the nature and pathogeny of which are essentially uncertain. Examples of the kind are the paraplegias observed after inflammatory affections of the kidney (Rayer, Stokes, Leudet), or lesions of the uterus and intestine (Brown-Séquard); the hemiplegia observed in pneumonia; the amaurosis following bruising of the frontal nerve; the paralysis of the arm observed after gunshot wounds of the thigh (Weir Mitchell, Morehouse and Keen), and the cases of so-called reflex inflammation (Brown-Séquard), etc.

Having found vaso-constriction of the vessels of the spinal pia mater in the course of experiments in which he ligatured all the nerves and vessels of one kidney, Brown-Séquard thought that he was justified in supposing that in a certain number of the clinical cases mentioned above a similar constriction of the spinal vessels occurred, causing weakness and even complete cessation of the functions of the region thus rendered anæmic.

Vulpian judiciously discussed all these cases in one of his lectures on the vaso-motor system. He showed that in several instances of urinary paraplegia obvious lesions of the nerve centres had passed unnoticed. With regard to the two cases in which Weir Mitchell [1] had seen a gunshot wound of the lower limb cause a paralysis of the corresponding upper limb, he asked if it was possible to eliminate with certainty any fall or traumatism whatever in the case of the upper limb. He admitted the existence of a material change in the brain in pneumonic hemiplegia. This last conclusion had already been arrived at by R. Lépine (219), who showed that pneumonic hemiplegias did not form a homogeneous group and that some of them were caused by cerebral ischæmia. The same conclusion was also arrived at in researches made by one of us in collabora-

[1] During the War of Secession a service of 400 beds for nervous disorders was given to Weir Mitchell and Morehouse, with W. Keen as their resident surgeon. The cases related by these writers include some which may be compared with those which we are observing at the present time. Such was the case of the man (Case XXXII [116]) who had been bruised on the arm by a shell fragment at the battle of Chancellorsville on May 3, 1863, and a fortnight later developed "a tonic spasm of the palmaris longus, brachialis anticus and flexor carpi radialis, causing a considerable flexion of the wrist." These phenomena progressively improved under treatment, and the wounded man was able to re-enlist in the New Jersey Volunteers. But as the result of fatigue the contracture returned. At the same time it was noted that the hand was always cold (the patient *never* left off wearing a glove) and emitted a markedly acid odour. Tenotomy was performed in June 1864, and in July, when the account ends, there was obvious improvement, but recovery was not complete. Although he devoted special attention to the study of reflex paralysis, which he called "paralysis from peripheral irritation," Weir Mitchell only exceptionally related cases of this kind. The cases which he studied under this name were examples of paralysis occurring at a distance from the point injured in another limb, and cases of convulsions or delirium. Under the influence of Brown-Séquard's ideas, which he nevertheless opposed, he thought that a group of anatomical elements in the spinal cord might be rendered inactive as the result of a lesion situated in a region of the body which had no direct relation with these elements. The term "reflex paralysis" or "paralysis from peripheral irritation" was obviously used at that time for the most heterogeneous cases.

tion with C. Lesieur (225) relating to several cases of pneumonic hemiplegia in which the presence of pneumococci was found in the cerebro-spinal fluid.

We must accept unreservedly the conclusion arrived at by Vulpian, but it should be specified that it in no way applies to the syndrome considered in his work, but merely to the group of so-called reflex paralyses which has just been criticised. "To sum up," says Vulpian, "in a review of the group of so-called reflex paralyses it will be found that it includes a larger number of cases in which there is a material lesion of the nerve centres, nerves or muscles, while among the remainder there are some which should probably be classified among neurotic paralyses."

This conclusion was formulated in 1875, and yet in 1886 in his lectures on diseases of the spinal cord Vulpian devoted a whole chapter to the study of the so-called reflex muscular atrophies. The fact is, that he did not in any way regard these nervous disorders as the same as those which he had previously criticised and which on this occasion he did not even mention.

It is nevertheless true that the exaggerated vogue which the conception of reflex nervous disorders enjoyed at a certain period was followed by a reaction, and disqualified in some minds conclusions based on incontestable facts.

The discredit into which the old views of so-called reflex contracture and paralysis of articular or abarticular origin had fallen is also explained by another cause, viz. the exaggerated development of the idea of hystero-traumatism.

As we have already said, there had been a tendency to attribute to hysteria almost all the nervous disorders following traumatism and not due to a definite lesion of the nervous system.

This is what A. Pitres wrote in 1891 in his Clinical Lectures on Hysteria *à propos* of a patient suffering from muscular atrophy following traumatism: "The words 'reflex atrophy' do not correspond to any definite nosological conception. The name is used

to-day to describe a certain number of muscular atrophies following traumatism. But a few years ago it was the practice to give the name of 'reflex contractures,' 'reflex paralysis' and 'reflex tremors' to a large number of disorders which really belong to hysteria and which naturally joined the group of hystero-traumatic disorders as soon as it was invented by the genius of Charcot. It will doubtless be the same with a large number of the cases of atrophy."

This point of view was due to the formerly orthodox doctrine according to which hysteria was capable of anything, including exaggeration of the tendon reflexes, and vaso-motor and trophic phenomena.

It must be admitted that with the old conception of hysteria distinction between reflex nervous disorders and hysterical phenomena was practically impossible, and confusion was inevitable.

The modern conception of hysteria, on the other hand, led to the separation of these two groups, and sometimes, though not invariably, furnished means for differentiating them. On what symptoms, then, was it possible to rely in order to establish this diagnosis?

Amyotrophy of reflex origin, when very pronounced, as it often is, could not be confounded with hysterical muscular atrophy which is of slight degree, but this is not always a sufficiently characteristic symptom to be decisive.

Exaggeration of the tendon reflexes when definite enabled one to exclude hysteria, but it is sometimes ill-marked and doubtful, and may even be absent altogether.

Vaso-motor and thermal symptoms might also serve as criteria. As long ago as 1904, in a lecture devoted to the objective symptoms which the will is incapable of reproducing (33), one of us expressed himself as follows.: "Trophic, circulatory and secretory disorders supply important elements of diagnosis. . . . If, for example, in a case of monoplegia, monoparesis or neuralgia in one of the lower limbs you find that the affected limb is colder than the other, that the secretion

of sweat is more abundant, and that the skin has a violet-red coloration, you may be sure that you are dealing with an organic affection."

In our first communications on reflex contractures and paralyses we emphasised again the importance of this fundamental characteristic, and we wrote (122): "We must admit that even in cases in which exaggeration of the reflexes was doubtful we had the impression that we were dealing with organic conditions, our impression being founded on the presence of local hypothermia." In the course of a discussion at the Neurological Society in October 7, 1915, one of us also made the following remarks: "I do not deny that a pithiatic paralysis may cause hypothermia or thermo-asymmetry when the paralysis is unilateral by reason of the lost or impaired functional activity, but the disorder in such cases is only slight. I am of opinion that a very pronounced and obstinate hypothermia is the sign of a more deep-seated perturbation. It is well known how frequent hypothermia is in organic hemiplegia, poliomyelitis and neuritis, but it seems to me that enough attention has not been called to the frequency and intensity of vaso-motor symptoms and hypothermia, which often develop after traumatic lesions and form part of the group of so-called reflex phenomena. These symptoms are common in cases of lesions due to gunshot wounds, even when these lesions have not caused much destruction of tissue and the nerve trunks and arteries have not been involved."

It should be stated, however, that the majority of neurologists did not share this opinion at that time.

Thus in a communication to the Neurological Society on July 1, 1915, to which we have already referred, Gilbert Ballet (82) demonstrated a patient with contracture of the right foot in a position of equino-varus, and said: "The forced adduction of the foot took place under the following conditions—

"On August 6, Lad . . . was operated on for hallux valgus by resection of the head of the first metatarsal. Three weeks later, when the operation wound had

cicatrised, the patient saw his foot assume a position of adduction, and the contracture fairly rapidly increased to the degree which you see at present. The patient was put under chloroform, and his foot immobilised in a plaster apparatus. In a few days' time, after the apparatus had been removed, the limb resumed its vicious attitude, and the dorsum of the foot showed a very marked œdema with coldness of the right foot as compared with the left." "This is obviously a case," Ballet concluded, "of a contracture of psychical origin with only a distant and indirect relation to the operation, which, as I may remind you, was performed on the first metatarsal, whereas the contracture involves the muscles of the tibiotarsal articulation. . . . This, then, is a contracture of psychical origin with œdema and associated vaso-motor disorders."

It should be noted that though this was an example of a contracture following traumatism and accompanied by vaso-motor symptoms, œdema and hypothermia, G. Ballet did not discuss or even consider the hypothesis of a reflex contracture.

This was not an isolated case; we have chosen it on purpose, because it was recorded by an eminent neurologist, to whose memory we are pleased to pay homage. It shows how much the principles established by Charcot and Vulpian had been forgotten.

Such were the ideas which were still in vogue a year ago.

New Ideas acquired by the Study of War Cases

We were induced to take up the study of reflex disorders and to add some observations of our own under the following circumstances. Our attention had first of all been directed to this study by the following case: In August 1915 a soldier came to our out-patient department at La Pitié suffering from a very marked limp with outward rotation of the foot following a wound of the upper and outer part of the right thigh. We found muscular atrophy of the thigh, without

Plate I

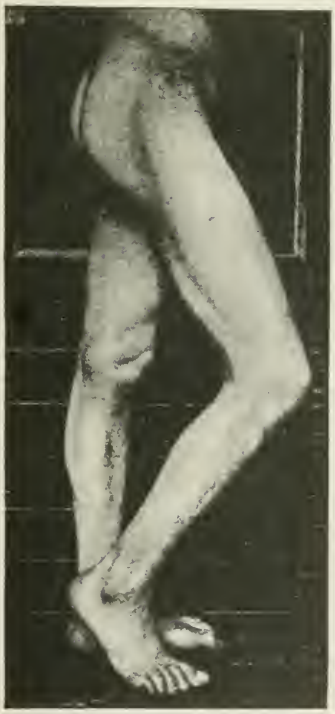
A. Contracture of the flexors of the leg on the thigh (126), following a wound of the middle third of the thigh (November 1914), without exaggeration of the knee jerk and without definite vaso-motor symptoms. The contracture persisted till a late stage of chloroform narcosis, and then completely disappeared. Its reappearance definitely preceded the return of sensation, cutaneous reflexes and conjunctival reflex, and was complete twenty minutes at least before consciousness returned (*v.* Appendix, Case XII, p. 665).

B. Same patient standing.

C. Contracture of the pelvi-trochanteric muscles with paresis of the foot and toes, following a shrapnel wound of the upper and outer part of the right thigh (September 1914). The right lower limb is in very pronounced external rotation; in a full-face skiagram the head of the femur appears in profile and completely hides the anatomical neck. Movements of internal rotation and of flexion of the thigh on the pelvis are slightly limited owing to the contracture and a slight fibrous retraction which was shown by examination under an anæsthetic. There is no lesion of the coxo-femoral joint.

The right knee jerk appears slightly stronger than the left in the waking state; during chloroform narcosis (122) an exaggeration of the tendon reflexes in the affected limb was found (September 1915), with right patellar clonus which could still be elicited an hour after complete recovery (*v.* Appendix, Case I, p. 660). We also found (May 1916) hypothermia and marked vaso-motor symptoms with reduction of the oscillations (4 in the affected limb as compared with 8 in the sound limb), considerable amyotrophy of the thigh (4 centimetres), mechanical hyperexcitability of the posterior tibial nerve, hypæsthesia of the affected foot with loss of cutaneous reflexes (the cutaneous reflex reappeared after warming) (145).

PLATE I



obvious disturbance of the electrical reactions. Examination of the hip showed a slight limitation of the movements of flexion and internal rotation of the thigh on the pelvis, which did not appear to be related to the motor disorder. The X-rays showed no lesion of the joint. The right knee jerk seemed to be a little more brisk than the left, but the asymmetry was doubtful. The tendo Achillis reflexes were normal and equal. There was no ankle or patellar clonus. On the other hand, the affected limb showed marked and persistent vaso-motor disturbance and a very definite hypothermia.

Owing to the severity of the vaso-motor disturbance we thought that we were justified in asserting that these symptoms were not due to hysteria and were probably connected with the so-called reflex phenomena. But the tendon reflexes in the affected limb were not remarkably exaggerated, and therefore the most characteristic sign was missing. There were reasons for supposing that the vicious attitude and muscular rigidity might be due to mere tendinous contractures.

To settle this question we thought it advisable to put the patient under chloroform. It was all the more justifiable in this particular case to employ any method likely to establish an exact diagnosis, as the patient had been considered by several medical men as guilty of exaggeration and even of simulation. Examination under chloroform showed the existence of a slight tendinous shortening, but it also enabled us to see that the vicious attitude and stiffness were due, in great part at least, to a contracture.

Moreover, during the stage in which the muscles were completely relaxed, after all the other tendon reflexes and the cutaneous reflexes were abolished, the affected side showed a very definite exaggeration of the tendon reflexes and even a patellar clonus which lasted for an hour after the patient had recovered from the anæsthetic (122).

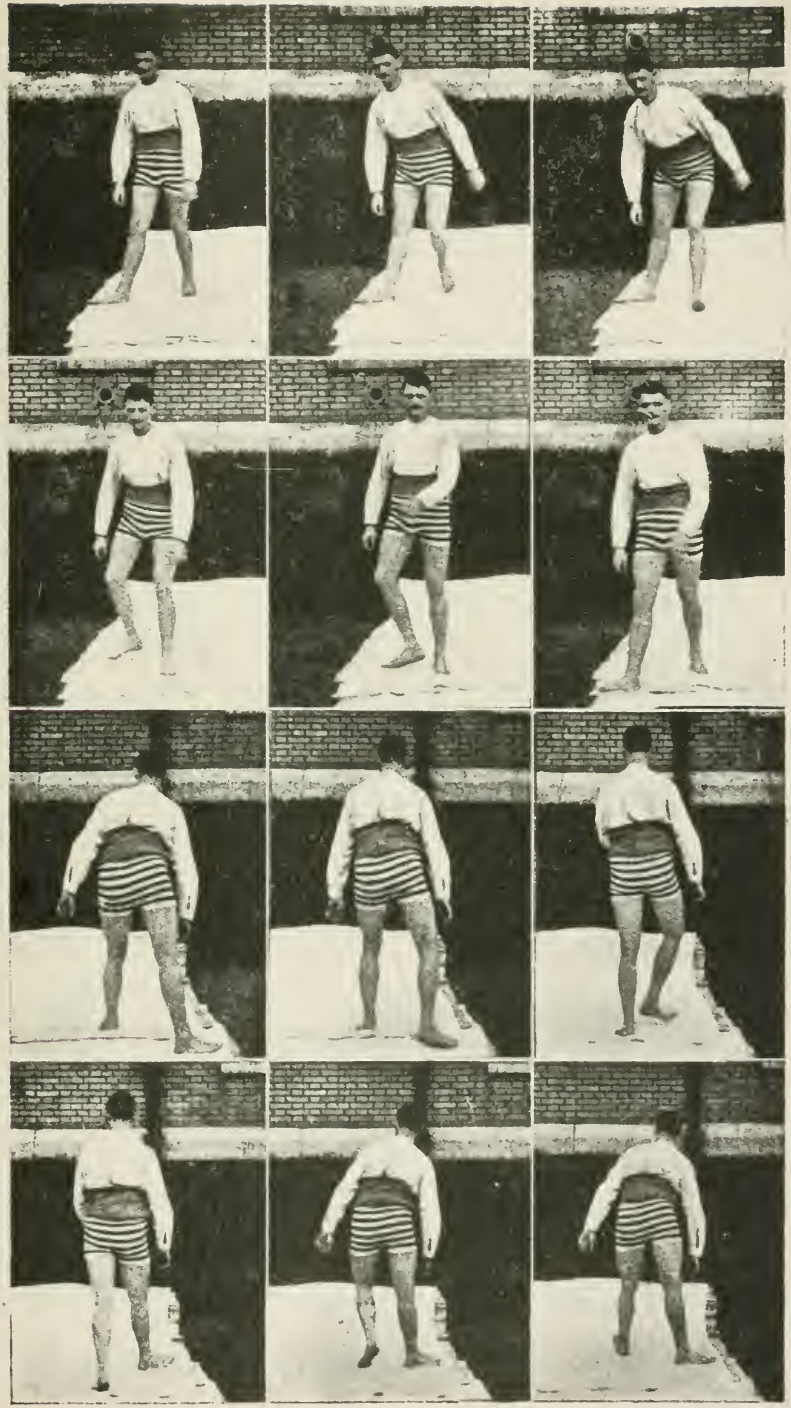
Since then we have examined under the same con-

Plate II

Cinematograph film of reflex claudication.—This man developed contracture of the pelvi-trochanteric muscles with paresis of the foot and toes after a wound in the right thigh. He presented a pronounced and obstinate claudication of a very peculiar type, but we have found most of its essential characters in other claudications of the same kind. A study of the different phases of each step (as seen from in front and from behind) shows the following peculiarities. When the two feet are resting on the ground, they are widely separated from one another; the right lower limb is also in very marked external rotation. When the affected foot moves forward, it is not carried directly forwards like the sound foot, but describes an arc of a circle with an internal convexity; it is first brought forwards and inwards by a movement of flexion of the leg which is then extended and carried forwards and outwards. While describing this movement the foot hangs in a position of equino-varus and sweeps the ground with its tip and outer border. Lastly, when the sound foot is raised from the ground and moves forward, the trunk is bent strongly to the affected side, and performs a very pronounced movement of salutation.

This cinematograph film (Gaumont) was shown on the screen at the Doulens Neurological Congress (January 1916).

PLATE II



ditions a certain number of wounded men presenting symptoms of the same kind, and we have several times noted *during narcosis this peculiar exaggeration of the tendon reflexes in the affected limb.*

We were thus placed in possession of a new symptom, or, at any rate, of a method which enabled us to detect increased excitability of the tendon reflexes in cases in which such increase was doubtful under normal conditions.

In some of the patients the symptom which we have just described was absent, but others equally significant were found : *the contracture disappeared during deep anæsthesia only ;* it was still present at a period when the conjunctival reflex was abolished, and stimulation by pricking the sound limb did not cause any reaction ; the return of the contracture was simultaneous with that of the tendon reflexes, and sometimes preceded the first signs of returning consciousness by twenty to twenty-five minutes. Moreover, *the effort at reduction during complete anæsthesia and after disappearance of any conscious reaction produced a spasmodic movement* which exaggerated the vicious attitude ; this was particularly the case in several instances of flexor contracture of the leg (126).

After making careful investigations into the changes in the reflexes and muscular tonus at different periods of chloroform narcosis, we directed our attention to the reflex nervous disorders following traumatism of the limbs, which were so often misinterpreted at the beginning of the war (125).

They showed that a close examination should be made before attributing nervous symptoms to hystero-traumatism, and that the absence of an exaggeration of the tendon reflexes in the waking state by no means justified such a diagnosis.

At the time when we were pursuing these researches numerous cases of difficult interpretation presented themselves, and these gave rise to a series of publications.

H. Meige, Mme. A. Benisty and Mlle. Lévy (127)

gave the name of "congealed hand" to a puzzling form of functional impotence, and also drew attention to the existence in these cases of a peculiar psychopathic state which became grafted on an initial organic condition. Claude (128) described side by side with hysterical phenomena "some similar manifestations which it is very difficult to classify," consisting of painful contractures which were localised and very persistent. Were they hysterical or reflex contractures? "We think," he writes, "that the contracture is of reflex origin, being caused by a nervous lesion or a sort of defensive movement, and that this attitude has subsequently become fixed and rendered permanent by a psychological mechanism resembling that of hysterical manifestations." Sicard (129) gives the name of "acro-contractures" or "acromyotonus" of the upper limb to contractures of the hand in various positions which resist all treatment, are aggravated by massage and electricity, and show only a slow and partial improvement as the result of very gentle procedures. "The pathogeny of such contractures," he writes, "remains obscure. . . . Hysteria is not concerned in their production." We may also mention the publications of Duvernay (124), dealing with post-traumatic "psychopathic and reflex contractures," which present the same characters; of Léri and Roger (123), and of Ducosté (197), on contractures following lesions of the peripheral nerves.

The impression derived from all these studies is that in a large number of centres of military neurology observers have been struck by the frequency of certain conditions, such as fixed attitudes and contractures which differ from the usual run of cases and which it has been impossible to classify.

Are they examples of hysterical manifestations, or are they phenomena of another kind? Such is the question which interests all these writers, and which they answer in different ways. It is quite obvious that they have no conclusive criteria to support their impression. Most of them have been surprised by the

persistency of the symptoms and their great resistance to psychotherapeutic measures. Although some for this reason reject the hypothesis of hysteria, not one of them compares these cases with the descriptions given by earlier writers.

The study of a patient presenting a type of paralysis hitherto undescribed gave a new direction to our researches. The case was met of a man suffering from flaccid paralysis of the hand and fingers following

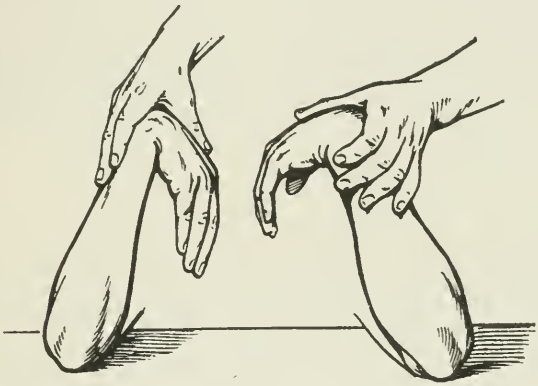


Fig. 4.—Hypotonus of the extensors of the hand following perforation of the hand by a bullet without lesion of the nerve trunks (*v.* Pl. IV, p. 524).

a bullet wound of the second dorsal interosseous space. Vaso-motor disorders and a pronounced hypothermia were found. There was a little diffuse and non-systematised atrophy of the muscles of the hand, forearm, and upper arm without R.D. The tendon reflexes on the affected side were preserved. The hypothesis of an organic affection of the centres or peripheral nerves had to be abandoned in the absence of any sign characteristic of such lesions. Were they, then, to be regarded as hysterical or simulated phenomena, as some had supposed?

On subjecting the patient to a methodical examination, in the search for some sign which would help us

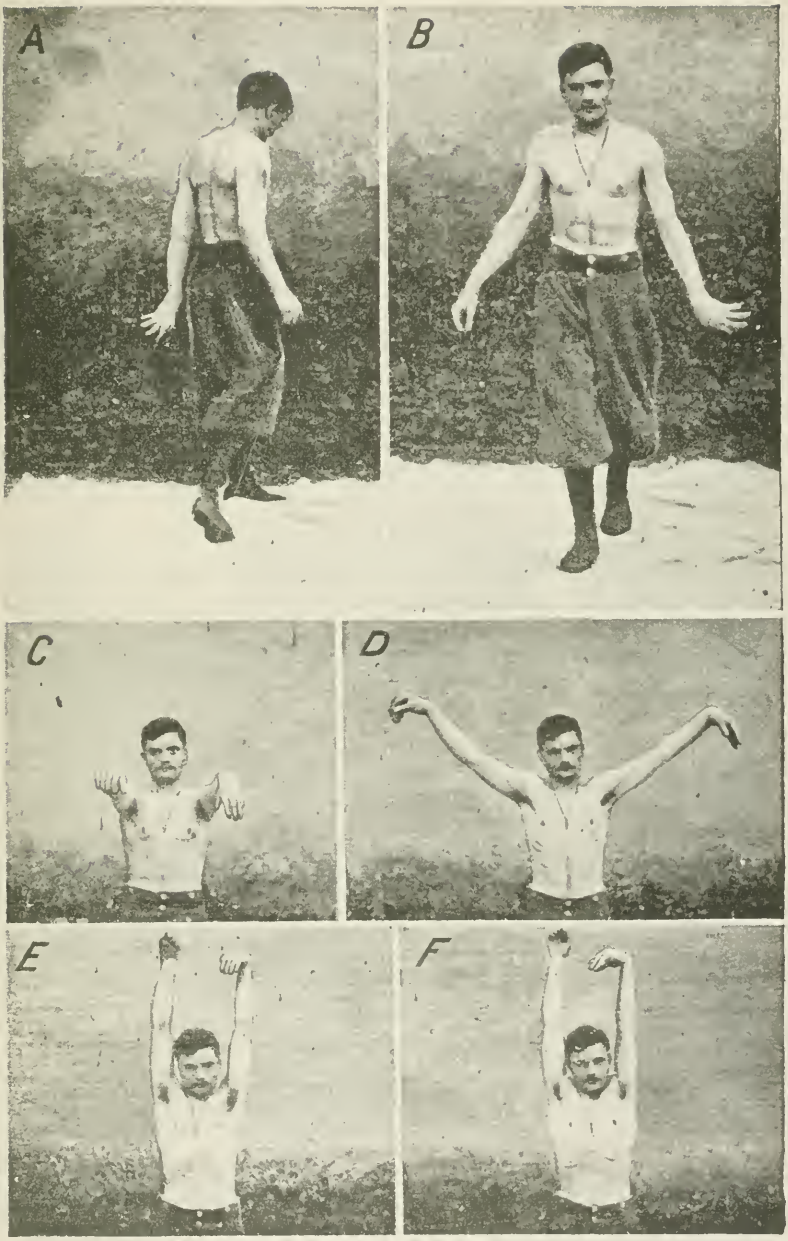
Plate III

Flaccid paralysis of the left hand and fingers with paresis of the movements of the forearm (131) which occurred five months after a wound of the second dorsal interosseous space (September 1914), and was preceded by a slight weakness of the hand. The bullet remained in the hand without perforating it, and was removed three months after the wound.

In January 1916 we also found vaso-motor symptoms and hypothermia and the existence of a very marked faradic, voltaic and mechanical hyperexcitability of the hand and forearm. Hypotonus is very marked and allows the hand to be hyperflexed on the forearm. When the patient moves the forearm or walks, the affected hand swings to and fro like a lifeless body, in every direction, as if it were obeying the laws of gravity only.

These peculiarities are clearly shown in the cinematograph pictures which we produce (Gaumont film shown at the Doulens Congress in January 1916). In May 1916 the condition of the patient who had been temporarily discharged and examined in his district by Porot (159) had not changed, and the signs mentioned above were still quite distinct.

PLATE III



to solve the problem, we were struck by the following symptoms : a remarkably intense *hypotonus* (especially of the wrist), which equalled if it did not actually exceed the hypotonus seen in paralysis following lesions of the deep nerves ; very marked *mechanical hyperexcitability* of the muscles of the hand and forearm, with *slowness of the muscular contraction*, and lastly *electrical hyperexcitability of the muscles with premature fusion of the faradic contractions* (131).

Objective signs which were independent of the will had thus been discovered, and consequently we felt convinced that these disorders were not due to hysteria.

On making a fresh examination of the patients whom we have already discussed, and in whom observation during anæsthesia had shown the undoubted existence of a disturbance of the spinal centres, we found the signs which we have just described, especially mechanical hyperexcitability with slowness of the contraction. They were also found in several patients who presented those peculiar types of contracture which had attracted the universal attention of neurologists (congealed hand, acro-contracture, and accoucheur's hand).

The existence of the mechanical hyperexcitability of muscles, described by us in this group of cases, has been confirmed by later observations made by Marie and Foix (132) and by Sicard (133).

On comparing those various groups of paralyses and contractures with one another, whose common attributes were that they were of traumatic origin and could not be attributed to an organic affection of the nervous system or to hysteria, we were led to establish a close connection between them. As some seemed undoubtedly to be due to a disturbance of the spinal centres, there were reasons for supposing that the others were of the same nature.

Lastly, we noticed that vaso-motor and thermal disorders usually occupied an important place in the symptomatology of all these cases, and that they were practically constant, although there might be differences

in their degree of intensity. We showed that the affected limb was abnormally sensitive to the influence of the atmospheric temperature, which implies a *local disturbance in the mechanism of vaso-motor and thermal control* (134).

Sphygmometric and oscillometric investigations, which were chiefly carried out in collaboration with Heitz (161), enabled us to detect the existence of a vascular spasm (135), which was specially marked towards the periphery. These phenomena had hitherto escaped attention.

We were also impressed by *the connection of vaso-motor and thermal disorders with disturbance of the mechanical excitability of muscles*, as shown by muscular hyperexcitability and slowness of the muscular contraction. These last modifications formed the object of a minute enquiry by means of the *graphic method* in collaboration with Hallion (153).

It is clear from the account which we have given that these various forms of contractures and paralyses, as to the nature of which there had at first been some doubt, are accompanied by objective signs of which we have endeavoured to estimate the value in our description. We have given them the name of "reflex" paralyses and contractures, because they resemble the amyotrophic or so-called reflex paralysis of which Charcot and Vulpian have given such an excellent description. Moreover, some of the phenomena which we have mentioned (observations during anæsthesia and character of the vaso-motor disturbances) supply new arguments in favour of the "reflex" pathogeny. In any case, our researches prove that there undoubtedly exists a *group of motor disorders of physiopathic nature*, which must be distinguished both from organic phenomena properly so called and from hysterical or pithiatic phenomena.

The pathological topic which we are studying formed the object of a general discussion at a special meeting of the Neurological Society on April 6, 1916, following

a report made by one of us dealing with the characters of so-called functional motor disorders. The neurologists present accepted the essential parts of our opinion, as is shown by the conclusions adopted unanimously in closing the discussion (140) :—

" *Regarding the matter exclusively from the standpoint of a medical board we have to distinguish among so-called functional motor disorders* (i. e. *motor disorders with none of the objective signs characteristic of organic affections of the brain, cord, or nerves, or of vascular lesions*)—

" 1. *Hysterical or pithiatic disorders properly so called.*

" 2. *Nervous disorders quite distinct from the preceding which are associated with real physiological disturbances of which the mechanism is still a matter of discussion, but which may be grouped with the reflex disorders observed after osteo-arthritic lesions.*"

Since the general discussion on nervous disorders of this kind at the Neurological Society, a certain number of articles have been published, which have confirmed our opinion, at any rate in its essential features. We will now give a short abstract of them:

In an article entitled " Reflex paralyses and reflex trophic disorders following wounds of the extremities; a contribution to the study of ascending neuralgia and neuritis," H. Gougerot and Albert Charpentier (151) express themselves as follows : " To sum up, from the first we have been impressed by the contrast between the localisation of the wound, with its apparently mild character and the importance, diffusion and ' ascent ' of the paralytic and very frequently trophic disorders; by the frequency of ascending pain, which is sometimes intolerable; by the distressing persistency of the pain, paralysis, and trophic disorders; their progressive aggravation many months after the wound; in short, by the gravity of the prognosis. . . . Such are the facts which the neurology of war has taught us; the recent publication of Babinski and Froment summarising their previous work (with which we were not acquainted) has induced us to publish these cases now, for they are similar to theirs. Not only do they

confirm them, but they add certain details, and illustrate the frequency of such cases."

After having discussed and dismissed the diagnosis of ascending neuritis (*v.* p. 586) H. Gougerot and A. Charpentier admit that these cases are examples of "a diffuse spinal and sympathetic reflex action."

"In our opinion," they add, "the pathogenic idea, as Babinski and Froment call it, matters little; the important thing is to be acquainted with these cases, the gravity of their prognosis, their obstinate character, and the frequent occurrence of ascending and reflex pain which make them resemble ascending neuritis and render the life of some wounded men so painful; in short, to realise that these patients are above all organic cases. In fact, many of them, as their wounds had been cured for two or three months, a year and sometimes more, had been considered as suffering from hysterical paralysis, and sometimes even as simulators."

Tinel (154), in a work entitled '*Wounds of Nerves*,' devotes a chapter to the study of so-called reflex contractures, though his opinion differs from ours as to their pathogeny. But he clearly distinguishes them from hysterical contractures as follows: "A large number of apparently paradoxical contractures are found, usually following slight traumatism, which one might be inclined to regard as mere hysterical contractures. But on close examination one finds the presence of a certain number of characters revealing the existence of an organic lesion, such as cyanosis, coldness, profuse sweating, trophic disorders of the skin and nails, fairly definitely systematised sensory troubles, and slight modifications of the electrical reactions and mechanical excitability of the muscles. These disorders have been carefully studied by Babinski and Froment, who have clearly distinguished them from mere functional contractures by the denomination of 'reflex contractures.'"

Porot (159), in an article devoted to "reflex manifestations (motor, vaso-motor and trophic) following disarticulation of the fingers," describes a series of cases closely resembling those which we have studied (134).

"We are simply dealing," he says, "with a special

case of motor, vaso-motor and trophic disorders of reflex character whose presence and persistence in the lower limbs are attracting increasing attention.

"The clinical reality and relative frequency of these disorders are no longer disputed nowadays. The description given of them in this journal by Babinski and Froment has shown us the principal symptoms of these reflex disorders which occupy an intermediate position between so-called hysterical manifestations and organic paralyses directly caused by affection of the peripheral nerves.

" We have been impressed by the relative frequency with which these reflex disorders occur in wounded men who merely had to undergo disarticulation or amputation of a finger.

" Out of a dozen cases of these reflex paralyses of the extremities which we had an opportunity of examining, we found four, or one-third of the cases, among men who had had one finger amputated. The initial wound had affected only one finger, which had to be disarticulated. There were no scars on the rest of the hand or on the foot, and the limb had not been wounded higher up.

" The clinical picture differed in each of these cases, and consisted in various combinations of motor, vaso-motor and trophic symptoms. All showed a mechanical and electrical hyperexcitability, and especially faradic hyperexcitability, which appeared to us to be the most constant feature."

A. Mézie (158), in a work entitled *Reflex Disorders and Cerebral Insufficiency*, also confirms our ideas both as regards symptomatology and pathogeny, but he adds " a physical or mental cerebral insufficiency " to the local factor.

In this connection we may remind our readers that Meige (127) had already emphasised the peculiar mental state of wounded soldiers suffering from the deformity which he described by the name of " the congealed hand." " One cannot help being impressed," he says, " by the general attitude of these patients. Silent and solitary, with anxious looks and few gestures, they appear to be perpetually on the alert. Their ' congealed hand ' seems to be the one object which occupies their mind."

We do not dispute the possible co-existence of a some-

what abnormal psychical state with reflex nervous disorders. But, on the one hand, we have seen a large number of patients of this kind who appeared to us to be quite normal mentally, and, on the other hand, obsession, anxiety and irritability, when they do exist in such cases, appear to be of secondary importance. Patients suffering from reflex nervous disorders may have been forcibly impressed by the persistency of the symptoms, the discomfort attending them, the failure of the therapeutical methods hitherto employed, and perhaps by the suspicion with which they were too frequently regarded.

In the course of a recent communication to the Neurological Society Souques, Megevand, Naiditch and Rathaus (160) showed a patient with so-called reflex paralysis of the lower limb. The following is a summary of the case, for which we are indebted to the authors: "This patient, after a superficial wound of the instep, has become affected with a motor disturbance which shows the principal characters attributed by Babinski and Froment to this kind of paralysis, viz. paresis of the whole limb, exaggeration of the tendon reflexes, loss of the plantar cutaneous reflexes, diffuse amyotrophy, increase of the idio-muscular and galvanic excitability, decalcification of bone, diminution of the amplitude of the oscillations with Pachon's instrument, and considerable hypothermia (5–10° C.).

"The immersion of the two feet in hot water diminishes the electrical and idio-muscular hyperexcitability on the affected side, causes the plantar cutaneous reflexes to reappear, and raises the temperature of the affected foot to the level of the sound foot and sometimes above it.

"We investigated the point as to whether normal subjects had a difference of temperature in symmetrical regions on the two sides of the body, and found that very often there was a difference which usually amounted to some fractions of a degree, but might be as much as a whole degree centigrade.

"We found that walking even when prolonged for an hour did not raise the temperature of the affected foot, and even lowered it a little, contrary to what occurred in the sound foot.

" Without entering into the pathogeny of so-called reflex paralysis, we are of opinion that genuine cases of this kind showed a collection of signs which separate them from hysterical paralysis and bring them into a close relationship with organic paralysis."

In a review on " Reflex Paralyses," which appeared in October 1916, Mirallié (162) expressed himself as follows : " At the beginning of the war this morbid type was confounded with hysteria, and at first all these cases were regarded as functional paralysis *sine materia*. As the result, however, of a more careful examination of these cases, the peculiar course of the disease, its characteristic symptoms and its resistance to psychotherapy, neurologists were led to place them in a special class quite distinct from pithiatic phenomena. Our knowledge of the subject is principally due to the work of Babinski and Froment, Meige, Claude, and Sicard." Mirallié reviews the symptoms of these disorders, and discusses their pathogeny, which he regards as complex, and on which he refuses to make a definite statement. One point at least appears to him to be quite certain, and that is the most important one : " All neurologists," he writes, " agree with Babinski in separating this condition from hysteria."

In a critical review of " The Psycho-neuroses of War," G. Roussy and J. Lhermitte (*Annales de Médecine*, September to October 1916) devote a chapter to the differential diagnosis of psychopathic contractures and make the following remarks *à propos* of reflex contractures :—

" The differential diagnosis between *psycho-neuropathic contractures* and *reflex contractures* to which Babinski and Froment have recently drawn attention is infinitely more embarrassing. These writers have made a careful study of reflex paralyses and contractures, and their description justifies the complete separation of such cases from similar manifestations of a psycho-neuropathic nature."

Lastly, in his Report made at the meeting of representatives of neurological centres, which took place on December 5, 1916, Grasset, though he classified these motor disorders among the neuroses, formed them into a special group.

"On superficial examination," he says, "these cases merely seem to be neuroses pure and simple. But—and this is characteristic of cases of this kind—on thorough examination specialists discover in these neurotic patients a variable number of symptoms described by Babinski and Froment, which *cannot be produced by the patient's will*, and which may be called the *minor traumatic signs* of the neuroses and psycho-neuroses of the second group."

CHAPTER VI

SYMPTOMATOLOGY

Reflex nervous disorders develop after wounds or various injuries of the limbs, and sometimes after frost-bite. In the case of wounds caused by projectiles, the lesion may only involve the soft parts and leave the skeleton and joints intact. The condition is often seen in cases of transfixion of the forearm, head, leg and foot, and it is interesting to note that the nervous disturbance usually extends above the area on which the trauma has been inflicted. We may add that the intensity of the nervous disturbance bears no relation to the duration of the infection, the extent of the lesion and the scar, or to the severity of the pain which these may cause.

It is rather difficult to state the precise moment at which the symptoms develop. They may appear immediately after the wound (Claude, Gougerot and Charpentier), but often the patient does not notice them until the dressing or splint has been removed. In other cases it is not until several weeks after the wound that the patient finds the limb assuming a vicious attitude, which progressively increases in spite of his efforts to prevent it. Sometimes the disorder, which was at first a mere paresis, becomes suddenly aggravated as the result of fatigue. Most patients with traumatic reflex contracture or paralysis were sent at first to surgical units, where they stayed for a varying period and were afterwards transferred to neurological centres at the base. In those at the front, therefore, there has hardly been any opportunity of seeing a case.

At the beginning of the last chapter we gave a short account of the different elements of the syndrome in

question. We shall now discuss the subject in greater detail.

Contractures and Paralyses: Clinical Forms

Clinically, reflex motor disorders may adopt various forms, such as contracture, paralysis, hypotonus, paresis or mere weakness, and frequently a combination of pareses and contractures. But, whatever may be its form, the motor trouble is usually incomplete, partial and limited; it shows a tendency to predominate in one segment of a limb, especially at its extremity, and thereby differs from pithiatic paralysis or contracture, which is generally more extensive and often complete and in which all the segments of the limb are usually affected.

It is possible to isolate some striking clinical types from among these varieties. Two patients corresponding to the same type may be as exactly alike as are two individuals presenting the same form of myopathy.

Contractures and Paralyses of the Lower Limb

Amyotrophic paralysis of the quadriceps.—This is the classical form of reflex atrophy which generally follows a femoro-tibial arthritis, but it may also be observed after wounds of the thigh or in the region of the knee in the absence of any lesion of the joint or patellar tendon (*v.* Fig. 6). The atrophy is sometimes associated with an obstinate paralysis of the quadriceps, and may be accompanied by extreme hypotonus (*v.* Fig. 15); usually, but not invariably, the knee jerk is exaggerated.

Contracture of the pelvi-trochanteric muscles associated with paresis of the foot.—This variety of motor disturbance is almost always found after traumatism in the region of the hip. In the dorsal decubitus the lower limb is in a position of very pronounced external rotation; the outer border of the foot being in contact

with the surface of the bed, passive movements of the thigh are somewhat limited, especially movements of internal rotation, and pull the pelvis round more

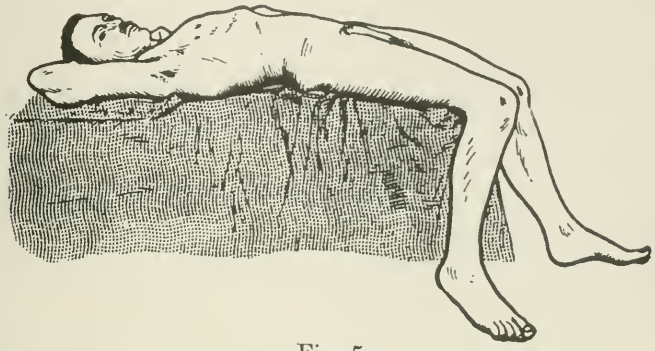


Fig. 5.

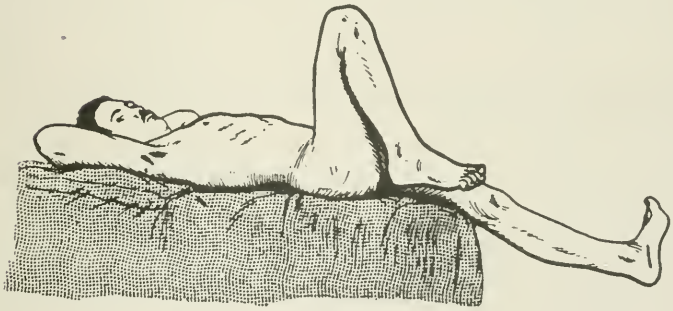


Fig. 6.

FIGS. 5 and 6.—AMYOTROPHIC PARALYSIS OF THE RIGHT QUADRICEPS ASSOCIATED WITH PARESIS OF THE FOOT, following a wound (November 4, 1914) of the outer aspect of the knee without lesions of the joint or patellar tendon. The reflex paralysis is accompanied (June 1916) by the following signs: pronounced and obstinate vaso-motor and thermal disorders with small ulcers of the foot, mechanical and faradic hyperexcitability of the quadriceps and muscles of the antero-external region of the leg, and hyperexcitability of the small muscles of the foot. The amyotrophy of the thigh is considerable, and the hypotonus is marked. The paralysis is very pronounced. When the patient is in the dorsal decubitus, with the legs hanging over the side of the bed (Fig. 5), and tries to extend them, he succeeds on the sound side only. In the affected limb (Fig. 6) the thigh is extended, but the leg, instead of being extended, becomes flexed.

rapidly than those carried out in the sound limb. This stiffness is due to a contracture of the pelvi-trochanteric muscles with which may be associated some fibrous retraction.

This contracture of the hip may be accompanied by paresis of the foot; active movements of plantar flexion, and especially those of dorsal flexion, are weak and of very small extent; it is the same with movements of the toes. When the paresis is less marked, the patient executes fairly extensive movements of dorsal flexion, but he lifts chiefly the inner border of the foot, which assumes a varus attitude. (*v.* Pl. VIII, p. 578).

These motor disorders almost always cause a very pronounced and obstinate claudication; sometimes a special complex type of limping occurs, to which we have already alluded. We will now study the different stages of each step; the patient, on starting to walk, having the affected lower limb behind the sound one and the feet widely separated. When he begins to walk he makes the affected foot describe an arc of a circle with an internal convexity, bringing it first forwards and inwards by a movement of flexion of the leg; then, extending the leg, he carries the foot forwards and very much outwards. While describing this movement the foot is hanging in a position of equino-varus, the toes and outer border being drawn along the ground. Lastly, when the patient raises the sound foot from the ground, and carries it forwards, the trunk bends strongly to the affected side, executing a movement of salutation, and as the body is then supported on the affected leg, a movement of torsion of the foot takes place which accentuates the varus attitude (*v.* Pl. II, p. 504).

In this form of contracture the knee jerk is not always definitely exaggerated in the waking state, but chloroform narcosis usually reveals a latent exaggeration of the reflex. Vaso-motor symptoms and mechanical hyperexcitability are usually very marked in the leg and especially in the foot. It is not unusual

also to find hypæsthesia of the foot with absence of the cutaneous reflexes.

Flexion contracture of the leg.—This clinical type which is more frequent than the extension type is very common. It is almost always a sequel of wounds of the thigh and leg; it may also be a complication of paralysis due to lesions of the sciatic nerve.

In standing and walking the heel is raised, and only the front part of the foot rests upon the ground (*v.* Pl. I, p. 502).

In the dorsal decubitus, it is found that in addition to flexion of the leg there is some flexion and abduction of the thigh. Active movements are possible, but limited by the contracture. It is the same with passive movements, though these are more extensive. It is impossible to produce complete extension even by taking the patient off his guard.

In the ventral decubitus, even when it is very prolonged, the flexion deformity persists, and the leg presents only slight oscillations, as we were able to observe in patients who were kept in that position for more than an hour at a time. The same test was applied to a case of hysteria major in a state of hypnosis in which the same type of contracture had been produced, but the woman did not succeed in maintaining this attitude perfectly for so long: as the test continued and her fatigue increased, the leg executed more and more extensive oscillations towards the horizontal plane, and finally almost reached the surface of the bed. It may also be mentioned that in the case of reflex contracture the angle of flexion is not appreciably modified from one examination to another, even when there is an interval of several days between each observation.

Contractures and pareses of the foot. Club foot and claw toes.—Motor disorders confined to the foot and toes chiefly occur after wounds of the leg or foot. They assume various forms, such as *paresis of the extensors with equino-varus, claw toes from contracture of the flexors,* or a rarer type in which there is a coexistence

of *flaccid paralysis of the great toe with extension contracture of the other toes* (*v.* Pl. VIII, p. 578).

Sometimes the toes are drawn together like the fingers in the "accoucheur's hand."

In the paretic form extension of the great toe does not accompany dorsal flexion of the foot as in the normal condition; the toes are not extended in any stage of walking.

These motor disorders, especially the most frequent one, paresis of the flexors of the foot and toes, may be associated with the various types of contracture and paresis of the lower limb which we have previously described.

Contractures and Paralyses of the Upper Limb

Flexion and pronation contracture of the forearm with paresis of the extensors of the hand and contracture of the flexors.—This clinical type is frequently seen, either in a complete or incomplete form, after wounds of the upper arm and forearm. The hand with the fingers extended is flexed at a right angle on the forearm, which forms an acute angle with the upper arm and is in a position of pronation. The Z-shaped appearance, resulting from this combination of deformities, is very characteristic, the affected limb resembling the appearance of a dog's paws when it is "begging" (*v.* Pl. IV, p. 524). Contracture of the flexors of the hand is associated with paresis of the extensors, and the tendons of the flexor carpi radialis and palmaris longus stand out beneath the skin. This combination of contracture and paresis of the antagonists sometimes gives rise to subluxations. In one of our cases it was fairly easy to apply the palm of the hand to the anterior surface of the forearm (*v.* Fig. 7).

In conformity with the rule that in reflex paralysis and contractures the symptoms predominate at the extremity of the limb, in the form in question the

symptoms were more marked and persistent in the hand than in the forearm.

Lastly, we may note that these motor disorders often coexist in varying degrees with the different forms of contracture of the hand which we are going to study, especially the so-called accoucheur's hand.

Accoucheur's hand.—This form of contracture appears to be very frequent. It has been seen in every neurological centre following wounds of the forearm, hand and fingers (*v.* Pl. V, p. 526).

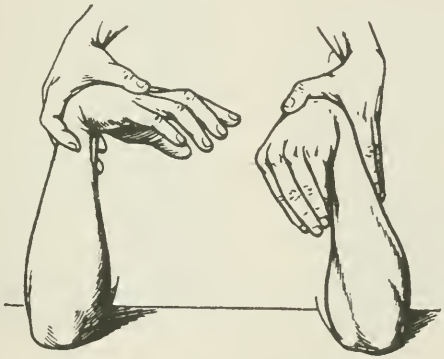


FIG. 7.—PARESIS OF THE EXTENSORS OF THE HAND ASSOCIATED WITH CONTRACTURE OF THE FLEXORS following perforation of the forearm by a bullet (August 1914). Subluxation of the wrist (April 1916) allowing the palm of the hand to be applied to the anterior surface of the forearm.

In this contracture the hand assumes a conical shape, and the fingers are arranged like the tiles of a roof, the ridge of the roof being represented by the middle finger alone or by the middle and ring fingers, which are either placed one above the other or both lying in the same plane. The index and little fingers which lie beneath the rest approach and sometimes even touch one another at their extremities, thus forming the sides of an equilateral triangle, the base of which is represented by the metacarpo-phalangeal joints. All the fingers are slightly flexed at the first and third phalanx, while the second phalanx is extended, sometimes even to excess, with a tendency

Plate IV

A. Contracture of the flexors of the hand and forearm with hypotonus of the extensors of the hand (attitude of dog begging) and accoucheur's hand. This man was wounded on August 22, 1914 (transfixion of arm at junction of upper and middle third); cicatrisation was rapid and did not take more than a fortnight. The deformity developed progressively after violent pain, affecting first the wrist and then the fingers; the patient tried, but in vain, to overcome it by raising his arm by means of splints. In October 1914 the elbow was bent to an angle of 45°, ever since then he has had systematic exercises, but improvement did not occur until November 1915.

In March 1916 extension of the forearm on the upper arm and extension of the hand or the forearm reach 120°. Flexion of the last phalanges and abduction of the fingers are possible, but these movements are carried out slowly and accompanied by tremors. The fingers progressively resume their original attitude, as soon as the patient makes no effort to keep them apart. Cold is ill borne, as is also excessive heat, which causes pain and twitchings of the muscles. The tendon reflexes of the affected limb are somewhat brisk. There is mechanical hyperexcitability of the biceps and small muscles of the hand.

B. Contracture of the flexors of the hand and forearm with hypotonus of the extensors of the hand and fibro-tendinous retractions. — Vaso-motor symptoms and marked hypothermia. Slight mechanical hyperexcitability of the muscles of the hand. Atrophy of all the tissues of the fingers. The symptoms developed after wounds of the lower part of the left arm and hand followed by amputation of the index (May 1915). Loss of power is not complete, the paresis is most marked in the hand (August 1916).

C. Contracture of the flexors of the hand with hypotonus of the extensors and subluxation of the wrist.— This shows the deformity known as accoucheur's hand with crowding of the fingers. The phenomena developed progressively after perforation of the forearm by a bullet, at the junction of the lower and middle third, complicated by fracture of the ulna (August 1914). The wound suppurated for three weeks, and immobilisation in a plaster apparatus lasted twenty-five days.

The loss of power is not complete (April 1916), the patient being able to carry out slight movements of flexion of the wrist; the affected limb shows tremor during an effort. The hypotonus is so marked that the hand can be applied to the anterior surface of the forearm (*v.* Fig. 7, p.523). There is also slight hypothermia of the hand, atrophy of the fingers and mechanical excitability of the biceps and supinator longus.

PLATE IV

to subluxation. The outline of each finger thus resembles that of a bill-headed cane.

The thumb is usually applied closely to the hand by its internal border; the first phalanx is most frequently strongly flexed; the last phalanx is extended or is even in a state of hyperextension, and its extremity fits into the groove formed by the other fingers. We may add that the little finger sometimes undergoes a movement of rotation inwards, so that the pulp of its last phalanx faces that of the thumb. The thenar and hypothenar eminences come close together, and sometimes enter into contact; the hand becomes hollowed out like a holy-water vessel, or shuts up like a book. Lastly, there is sometimes a certain degree of adduction or abduction of the hand.

These deformities are more or less marked in different cases; sometimes there is only a faint indication of them. We may meet with the typical accoucheur's hand, which is extremely frequent, or hypertonic conditions resembling it, and almost always reproducing some of the features which we have just described, such as the hollow hand, forced adduction of the thumb, overriding of the fingers, arrangement like the tiles of a roof, and position of the fingers like a bill-headed cane.

Which are the muscles whose contracture produces this deformity? There is every reason to suppose that the principal part is played by the adductor pollicis and interossei, especially the adductors or palmar interossei. These muscles are innervated by the ulnar, but it should be noted that the deformity is quite different from that resulting from lesions of this nerve.

This contracture has a special peculiarity apart from its form. An attempt to correct the vicious attitude succeeds easily; the hand when left to itself may retain its normal shape for a few minutes, but it gradually resumes its previous one, as if it had been compelled to by its muscular tonicity. This variety of contracture is distinguished from contractures

Plate V

A. Hypertonic condition with the deformity known as "accoucheur's hand."—Contracture of the hand following perforation of the middle of the left forearm by a bullet, complicated by fracture (February 1915). With the reflex motor disorders ("congealed hand," paresis and limitation of movements) was associated a lesion of the median nerve with R.D. of the muscles of the thenar eminence (March 1915). In October 1916 only a slight diminution of faradic excitability remains, but the reflex motor disorders have not yet undergone much change in spite of regular treatment (electricity, mobilisation and re-education).

B. Contracture of the hand, which appeared after perforation of the wrist with fracture of the os magnum (November 1914).—The ring and middle fingers override the little finger and index, which are in an attitude of forced adduction with their last phalanges in contact. The loss of power is not complete (June 1916); the patient can bring his fingers into the same plane by extending them, but they resume their original position as soon as he attempts to flex them; in the case of the middle finger, flexion of the last phalanx only is possible, the other phalanges remain extended (a similar motor dissociation cannot be produced on the normal side). Slight but obstinate vaso-motor symptoms and hypothermia. Mechanical hyperexcitability of the biceps and supinator longus.

C. Contracture of the hand following a bullet wound of the forearm complicated by fracture of the ulna (August 1914), one of the most pronounced types of the "accoucheur's hand" deformity. The hand is, as it were, folded longitudinally, the thenar and hypothenar eminences enter into contact, the contracture of the adductor is very marked, the fingers override one another, and have the bill-headed cane appearance. The hypotonus of the flexors of the fingers is very pronounced and allows subluxation of the phalanges (*v.* Fig. 9, p. 555). There are vaso-motor and thermal disorders, and a very definite atrophy of all the tissues of the fingers (December 1915).

D. Same patient as A (dorsal surface of affected hand).

E. Contracture of the hand following a shell wound with fracture of the bones of the forearm (November 1914). The loss of power is not complete (March 1916), the patient can separate his fingers, but as soon as he makes no further effort to keep them apart, they assume slowly and progressively their original position. All the movements of the hand are easily carried out.

PLATE V

previously described and from contractures of the lower limb, which are usually very pronounced and rather difficult to reduce. In order to mark this distinction it may be justifiable to use the term "hypertonus," which has also been employed by Sicard, Meige, and P. Marie and Foix.

Contractures of the hand of various types.—Contracture of the hand may also give rise to other deformities, such as *hyperextension and abduction of the fingers combined with flexion of the wrist* or hand like a circumflex accent (*v.* Pl. VII, p. 572), *extension contracture of the fingers and hand*, and *clawhand with crowded fingers* (the index and ring fingers lying on the middle finger) (*v.* Pl. VI, p. 564).

Hypotonic paralyses or pareses of the hand and fingers.—Simple paresis of the hand and fingers is not rare. Complete paralysis appears to be exceptional. In such cases there is a contrast between the absolute or almost absolute loss of power in the extremity and the relative freedom of the movements of the forearm on the upper arm and of the upper arm on the shoulder. The most striking feature in this clinical type is the extreme flaccidity of the hand, which swings to and fro during walking in a lifeless manner, even when the arm remains still and is kept close to the side. In other words, hypotonus in such cases is very pronounced (*v.* Pl. III, p. 508).

The deformities caused by reflex contractures of the upper limb show a striking clinical resemblance to *tetany*, apart from the paroxysmal character of this affection.

Some of the terms recently employed emphasise this resemblance of the symptoms, acro-contracture being synonymous with "contracture of the extremities," an expression formerly used to designate tetany. In like manner, the term "accoucheur's hand" was employed by Trousseau to indicate the form presented by the hand, of which he gave such a remarkable description: "In the upper limbs the thumb is forcibly and violently adducted, the fingers

are pressed close together and semi-flexed over the thumb, the moment of flexion taking place, as a rule, only at the metacarpo-phalangeal joint; the palm then being made hollow by the approximation of its outer and inner borders, the hand assumes the shape of a cone or rather the shape given it by the accoucheur when he introduces it into the vagina." Does not this present a striking resemblance to the types of contracture which we have just studied?

The following passage relating to tetany is taken from Dejerine's work (64): "In his study of infantile tetany Escherich described extension and separation of the fingers, their arrangement like the tiles of a roof, with forced adduction as in arthritis deformans, the "scribe's hand" position, the frequent pronation of the hand with flexion to the ulnar border, the thumb inverted, and the fingers extended at the metacarpo-phalangeal joints and flexed at the distal joints. Then, if the contracture extends to the whole of the upper limb, the patient assumes an attitude resembling that of a dog on its hind legs (Escherich), the arms being kept close to the body, the forearms flexed and the hands pronated."

After reading these descriptions a glance at the annexed figures is sufficient to show that the deformities produced by reflex contractures exactly correspond to those of tetany.

Vaso-motor and Thermal Disorders

In the majority of cases vaso-motor and thermal disorders are very definite, especially in cold weather; this is a point which we would emphasise at once.[1]

The phenomena do not correspond to a well-defined nerve territory. They may occupy the whole of the

[1] This study of reflex vaso-motor and thermal disorders, including the tables on pp. 668 *et seq.*, has been chiefly taken from a recent work carried out in collaboration with J. Heitz ("Vaso-motor and Thermal Disorders in Reflex Paralyses or Contractures," Babinski, Froment and Heitz, *Annales de Médecine*, October 1916).

affected limb, but they predominate at the extremities in which they frequently persist. The affected hand or foot is cyanosed, mottled or of a uniform salmon-red tint. The slightest pressure causes a local ischæmia, and the white spot thus produced is slow in disappearing. This disturbance of the coloration of the skin is clearly shown in comparing it with that of the sound side. In the lower limb, the asymmetry in the coloration is more pronounced when the patient is standing than when he is lying down.

In addition to the change in colour, a slightly infiltrated appearance of the skin is fairly frequently met with, especially on the dorsal surface of the hand, which presents a "succulent" appearance.

Sometimes even there is a real œdema which pits on pressure, especially in the lower limb. The œdema may be localised in the dorsum of the foot, or in exceptional cases extend up to the knee. We may add that the consistency of this œdema is generally hard, and that the pitting on pressure is slight.

Hypothermia definitely perceptible to the touch and sometimes very pronounced is associated with the vasomotor disorders. The difference in temperature between the affected limb and the sound limb is as much as 8° or 8·5° C. in the most marked cases.[1] As a rule the hypothermia is most pronounced at the extremity (fingers, toes, hand or foot), and becomes progressively less from the extremity to the root of the limb. It is generally still found to be distinct at a level at which the skin has resumed its normal coloration. We may add that the hypothermia always passes appreciably beyond the level of the wound, *e. g.*, it may be perceptible at the knee and sometimes even on the thigh with a wound of the foot, or be found at the shoulder after perforation of the wrist. It may vary within certain limits from day to day, or even from one moment to another, according to the external temperature, or according as the patient has been

[1] The local and superficial temperature was taken with Mlle. Grunspan's thermo-electric pyrometer (210).

living in a warm room or not. After a short immersion in water at 100·4° F. or 104° F., the affected limb sometimes becomes as hot and even hotter than the sound limb; the white area produced by pressure of the finger on the back of the affected hand or foot instead of disappearing more slowly, fades quite as rapidly, and even more so, than on the affected side. Return to the previous temperature is usually more rapid in the sound limb.

The local temperature may also undergo further modifications due to other influences, *e.g.* exercise and electrical treatment. The rapid changes due to these causes have led some observers into error by making them suppose that certain cases had been cured, whereas there was only a temporary improvement.

As a matter of fact, when the patient is removed from the action of heat, the thermo-asymmetry usually returns quickly, and the hypothermia reappears in the affected limb, which is to a certain extent *heterothermal.*

What do we learn from examination of the vessels? Palpation sometimes shows *weakness of arterial pulsation* in the affected area. This phenomenon is particularly marked in the dorsalis pedis, the pulsation of which may not be perceptible, while it is normal on the opposite side.

The idea of an arterial lesion, such as compression or partial obliteration, may then suggest itself, but this impression is contradicted by the precise information afforded by the sphygmomanometer. Potain showed long ago that the arterial pressure could not be estimated by merely feeling the pulse.

Two methods were available for measuring the systolic pressure in our patients, viz. the oscillometric method of Marey and Pachon, and the method of Riva-Rocci and Vaquez.

We had to give up the former method. As is well known, it is based on the study of the oscillations caused by compression of the artery by an armlet distended with air.

Pachon regards as indicating the systolic pressure the point at which the oscillations, after being reduced to a minimum by strong compression, begin to increase definitely as the result of decompression.

We shall see later that in a limb affected with reflex disorders, the oscillations are almost always of reduced amplitude, and the difference between the two sides is sometimes very considerable; under these conditions it was very difficult to determine the exact moment at which the modification indicated by Pachon occurred.

The same objection did not apply to the method of Riva-Rocci and Vaquez, which is based on reappearance of the pulse below the armlet when the pressure is being gradually reduced. This was the method which we adopted in all our cases for measuring the arterial pressure, placing the armlet in the middle of the upper arm in the case of the upper limb, and immediately above the malleoli in the case of the lower limb. The systolic pressure is indicated in the former case by the return of the radial pulse, and in the latter by the return of the pulse in the dorsalis pedis or posterior tibial artery.

The figures which will be found in the tables in the Appendix (*v.* p. 668 *et seq.*) show that in the upper limb the systolic pressure is the same on the two sides, and that any difference between the two does not exceed 1 cm. Hg., as may be seen normally in some individuals. In the lower limb the investigation of the pulse in the dorsalis pedis or posterior tibial is a little more difficult; even in some normal subjects it may be difficult to feel on the two sides. When the calibre of the artery is narrowed, as frequently occurs in reflex paralysis, it is sometimes impossible to feel the return of the pulsations in the affected limb and consequently to measure the systolic pressure. After immersion in hot water, the arterial pulsations become much more definite, and the pressure in symmetrical arteries is then found to be equal (Babinski and Heitz).

The diastolic pressure was measured with Pachon's

oscillometer by reading the number corresponding to the last greatest oscillations in the course of decompression. It is not modified in reflex paralyses.[1]

We endeavoured to determine the *amplitude of the greatest oscillations* in our patients in the arm (brachial artery), thigh (femoral artery), wrist and instep. (A comparison of the figures indicated by the pointer of the manometer and that of the oscillometer shows that the greatest oscillations correspond to a pressure of 1 to 2 centimetres higher than the diastolic pressure.)

The greatest oscillations represent the greatest expansion possible in the case of a large artery, and the greatest expansion of the combined arterial trunks and branches, when the cuff is placed near the extremity. To measure them we noted the highest and lowest figures reached by the oscillation of the pointer on the scale of Pachon's apparatus, *e. g.*, 3° and 12°. Then we subtracted 3 from 12 = 9, which we multiplied by two (on account of the secondary line which divides each of these degrees on the scale of the oscillometer), which gives a total of 18 divisions covered by the pointer as the result of arterial pressure.

We may note, first of all, that individuals who have no lesions of the limbs or any paralysis do not show any important difference between one side and the other in the amplitude of their oscillations (Heitz). There may, however, be some asymmetry. But it is not very marked, and is generally more pronounced at the root of the limb than at the extremity. At least, this is the result of the observations which we have made hitherto and are still pursuing.

[1] Our method of procedure was as follows: Recklinghausen's cuff, adopted by Vaquez, measuring 12 cm. in width, was the only one used, and was attached to Pachon's oscillometer. The systolic pressure was taken, both in the upper and lower limb, by Riva-Rocci's method, and then without removing the cuff the diastolic pressure was taken in the same artery. We also took the maximum amplitudes of the oscillations, and with the same cuff took the same measurements of other arteries (femoral and radial), whenever it seemed necessary.

The following are some figures taken from *normal subjects*. In the brachial and femoral — 14° : 14°; 16° : 14°; 26° : 23°; 26° : 26°; 18° : 17°; 20° : 16°. In the radial and tibials in the same individuals—5° : 4°; 4° : 3°; 8° : 10°; 8° : 12°; 5° : 5°; 8° : 6°.

In wounded patients suffering from *reflex nervous disorders* the difference between the pressures on the two sides is much more pronounced, at least at the extremity of the limb. Thus in the brachial and femoral we found—7° (affected side) : 14° (sound side); 11° : 14°; 12° : 14°; 14° : 16°; 14° : 17°; 16° : 18°; 10° : 18°; 18° : 20°; 18° : 21°; 14° : 20°; 20° : 22°; 14° : 26°. In the radial and tibials we found—0·5° (affected side) : 6° (sound side); 1° : 5°; 1° : 7°; 1° : 8°; 1° : 12°; 2° : 6°; 2° : 9°; 2·5° : 10°; 4° : 8°; 6° : 12°; 7° : 14°. We have purposely chosen instances in which the symptoms were most marked.

The oscillations observed in the affected limb are frequently in the proportion of 1 to 2 or 1 to 4 in relation to those of the sound limb; in exceptional cases they may be in the proportion of 1 to 8 or even 1 to 12.

As a rule, then, there is a diminution of amplitude in the oscillations on the affected side, and this diminution is often considerable. But, if it occasionally happens that the difference does not exceed that which may be observed normally, it is always on the affected side that the lowest figure is found, and the difference is always greater towards the extremity of the limb.

This was just what we saw in the case of hypothermia, and it can readily be ascertained that the reduction in the oscillations goes hand in hand with the chilling of the skin; it is, therefore, like the latter, much more pronounced during the winter months. It was at this period of the year that we found the radial or tibial oscillations on the affected side sink to 0·5° or 1°, whereas on the sound side we found 4°, 6° and even 8°; it is at this time also that the reduction may become very appreciable even in the brachial or femoral.

On a hot day, on the contrary, it will often be possible not to meet with any local hypothermia, and to find that the oscillations have the same amplitude on the two sides.

For the purpose of studying the disturbance of the peripheral circulation in the fingers and toes we had recourse to Gaertner's tonometer, which was formerly employed for measuring the blood-pressure, but soon fell out of use when it was found that it measured the arterio-capillary tension only, which is too liable to undergo variations from different causes.

The following technique is employed in these investigations : a distensible rubber ring, 1 to 2 cm. wide, of which the internal pressure can be measured by a manometer, is applied to the finger. The last joint is rendered bloodless by tying a rubber band round it, the ring is distended to its maximum, and then the compression is gradually diminished until the blood returns to the finger, as is shown by the change in the colour of the tissues, which become pink again.

As a rule, the figure obtained by this process is either equal to that of the systolic pressure (Riva-Rocci-Vaquez method), or more frequently lower by a few centimetres; it is calculated that it roughly corresponds to the systolic pressure in the collateral arteries of the fingers (L. Gallavardin) (207).

The results which we obtained by this process agree with those which we derived from an examination of the oscillations; the figure obtained by Gaertner's apparatus is constantly lower in the fingers and toes on the affected side, it even seems that the fall of the arterio-capillary tension is more pronounced than the reduction of the oscillations at the wrist or malleoli. We found, in fact, the following figures—0 (affected side) : 3 (sound side); 0 : 9; 4 : 7; 5 : 10; 6 : 12; 9 : 15.

During the cold weather we fairly often observed that the blood failed to return, even when the pointer of the tonometer was at 0; a fact which indicated an extremely intense vaso-constriction. We may add

that the degree of the fall varied from one case to another, and even from one day to another in the same individual, under the influence of variations of temperature or any other cause which might act on the circulation of small vessels.

The vascular troubles observed in reflex paresis and contractures, increase, as we have said, under the influence of cold, and decrease and even disappear after immersion in hot water. The amplitude of the oscillations increases considerably in the affected limb as the result of heat; it tends to be equal to the amplitude of the oscillations on the sound side, and exceptionally it actually exceeds them. In this connection, it is important to state that a local cold bath, at least one of short duration, as in the test which we use with our patients, does not normally cause considerable modifications in the amplitude of the oscillations (*v.* Appendix, p. 696).

All these phenomena, which we have studied in some detail, indicate *a profound disturbance of the vaso-motor and thermal mechanism.*

We may note incidentally that *examination of the blood* taken from the finger on the affected side and compared with that of the finger on the sound side showed that it was *a little less rich in red cells* (20,000 to 50,000 red cells less per cubic millimetre in the most marked cases) and *hæmoglobin* (colorimetric method), and that the hæmoglobin was *less reduced* (spectroscopic method). These differences may diminish after warming. But our investigations as to this last point have not been sufficiently numerous to justify any conclusions.

CHAPTER VII

SYMPTOMATOLOGY (*continued*)

Mechanical Hyperexcitability of the Muscles. Slowness of the Muscular Contraction

Mechanical hyperexcitability of the muscles is in our opinion a very important sign, and was one of the first to strike us in the course of our investigations. It is best marked in the paralytic or paretic forms, but it is also found in the hypertonic varieties. The phenomenon is most commonly seen and is most definite in the small muscles of the extremities, but it is also found in the muscles of the leg and forearm and in the quadriceps. Even a slight percussion of the muscles causes a movement of great amplitude, which is often slow and sustained.

This mechanical hyperexcitability enables us to act upon each individual muscle separately with as much precision as with an electrical stimulus, and to obtain in a state of purity, so to speak, the movement corresponding to each. This percussion of various points of the thenar eminence may not unfrequently cause successive contraction of the abductor pollicis (extension of the first phalanx of the thumb and its opposition to the index), flexor brevis pollicis (opposition to the little finger), and adductor pollicis (flexion and adduction of the first phalanx of the thumb). Percussion of the hypothenar eminence produces a movement of abduction of the little finger with flexion of the first phalanx, extension of the last two, and a tendency to opposition to the thumb. Abduction of the index frequently associated with adduction of the thumb, like a crab's claw, is obtained by percussion of

the first interosseous space. Gentle percussion with a small reflex hammer, or merely with the handle of an ordinary hammer, can thus enable one to obtain the whole scale of movement, just as if one were testing successively the keys of a piano. When the phenomenon is well marked, it is very characteristic. As a rule, in the normal condition, it is possible to obtain only a faint rapid movement of feeble amplitude. It should also be noted that owing to individual differences mechanical hyperexcitability has most value when it is unilateral.

The contraction so obtained must be distinguished from a reflex movement; it occurs when the body of the muscle is struck, and does not take place when percussion is made on the tendons or bony surfaces. The expression "muscular reflex" is ambiguous, as there is no justification for supposing the existence of a reflex arc. There is every reason, on the contrary, for regarding this phenomenon as related to idio-muscular contraction; in cases in which it is ill developed mechanical hyperexcitability is shown by a sort of vermicular contraction without change of position in the corresponding segment.

The comparative study of the mechanical hyperexcitability of the muscles of the foot and exaggeration of the osso-tendinous reflexes, such as is often observed in organic hemiplegia or spinal paraplegia, enables one to distinguish between these two phenomena.

In the case of exaggeration of the tendon reflexes, on whatever part of the foot the percussion is made, whether the dorsum, plantar aspect, inner border, muscular surface, or, best of all, the bony surface, the movement of the toes is appreciably the same; the first phalanx is flexed, the rest are extended, and a slight movement of abduction occurs.

In mechanical hyperexcitability of the muscles, on the contrary, movement of the toes only takes place if the percussion is made on the muscles; it varies according to the nature of the muscle affected, which responds to the mechanical stimulus by a contraction

of the same form if not of the same intensity as that which would result from an electrical stimulation of the same point. Percussion of the postero-external part of the foot (extensor brevis digitorum) causes mere extension of the toes; of the anterior part of the metatarsus (interossei), flexion of the first phalanx combined with extension of the other phalanges and abduction of the toes; percussion of the internal border of the foot (adductor hallucis) causes adduction of the great toe, of the external border (abductor minimi digiti), abduction of the little toe, and of the sole near the arch of the foot (flexor brevis digitorum). flexion of all the phalanges. Percussion of the bony surfaces, such as the heel or the toes, is not followed by any movement.

Mechanical hyperexcitability of muscles is closely connected with hypothermia. When ill marked or doubtful, immersion of two symmetrical limbs for a few minutes in very cold water is often sufficient to make it obvious. The excitability in that case is often very considerably increased on the affected side in the muscles of the extremity, whereas on the sound side it is not appreciably modified as a rule; sometimes, however, we thought that in such patients the chilling of the sound limb increased the mechanical excitability more than in normal individuals, but the difference between the two sides was always very appreciable. The cold water test, therefore, can be used for diagnosis.

We may note in passing that the modifications of the muscular contractility produced by chilling are in every way comparable to those seen in the frog which has been plunged into iced water. The process in that case is doubtless more violent, and the chilling much more intense, but the result is similar. Slight percussion of the gastrocnemius which normally does not cause a definite contraction produces a slow, sustained and very ample extension of the frog's foot, a movement which disappears rapidly after immersion in warm water. This mechanical hyperexcitability often corresponds to a pronounced but transitory paretic state.

In wounded men suffering from reflex nervous disorders, after immersion of the affected limb in hot water, or after diathermy, the muscular contraction caused by percussion changes in character. It ceases to be prolonged, and becomes rapid and ill sustained. It may also show a considerable diminution; the asymmetry may even become hardly appreciable after the application of heat. It should be noted, however, that this diminution of muscular contractility is produced by a moderate rise of temperature only. Excessive heat may produce an exaggeration of the excitability of the muscles, which then respond to mechanical stimuli by extensive but very rapid contractions.

Some writers have held that in wounds of the limbs the slowness of the contraction indicated the existence of a nerve lesion, and that it was equivalent to the reaction of degeneration.

" Practitioners," wrote Pitres (206) in a recent report, " who have not at hand the electrical apparatus which is only to be found in a specialist's consulting-room or an electrical laboratory, can replace to a certain extent electrical diagnosis by mechanical diagnosis. As long as the contraction on percussion is rapid, they may be certain that the muscle is but little affected; when it becomes slow they may be sure that there is R.D."

We do not share this opinion. When there is a profound disturbance of the vaso-motor and thermal mechanism, and when the external temperature is low, the contraction in such cases is often remarkably slow; in fact, it is as slow as in many cases of definite reaction of degeneration. In order to establish a closer and more scientific parallel between these two classes of cases, we had recourse to the graphic method with the valuable collaboration of our colleague Dr. Hallion. The following is an account of our investigations (153).

Slowness of the Muscular Contraction obtained by Percussion in Reflex Contractures and Paralyses. Study by the Graphic Method

Technique.—The patient's forearm and hand are bandaged to a wooden splint, padded with wool, leaving free and uncovered either the thenar eminence and thumb or the hypothenar eminence and little finger, according to the case. The patient is seated, with the forearm resting on a table; a cushion forming an inclined plane is arranged so as to keep the hand elevated.

The movement to be studied is taken up by a Marey's collecting tambour and transmitted by a rubber tube to a recording tambour, which registers it on a revolving cylinder.

In a first series of experiments we made the cylinder revolve at top speed. But we found on examining the tracings that the prolonged contractions lasted too long to be recorded completely in one revolution of the cylinder. So to obtain a complete record of those contractions which terminated very gradually we decided to make a fresh series of examinations in three of our cases, in which we reduced the rate of the cylinder and gave the recording lever a greater length, so as to make the descending part of the curve more prominent, with the result that the tracings were reduced in breadth but increased in height. Lastly, we fastened the splint holding the limb to the table instead of merely using the hand to steady it.

The collecting tambour was provided with a system of joints, so as to follow the movements of the finger explored. The latter was connected with the lever of the drum by means of a rubber ring.

When everything was ready, one of us made sure of the immobility of the limb to be explored with one hand and with the other performed percussion according to the ordinary clinical method.

Each tracing was accompanied by a time-marking record.

We made several series of tests in succession on the same subject, varying the experimental conditions, and on several occasions made a comparative examination of the sound side and affected side under identical conditions.

In each case we examined the limb first at the atmospheric temperature, and then after treatment with hot or cold water. The application of cold was particularly necessary under the circumstances. It should be noted that all the tracings were taken between May 26 and June 27. The temperature of the atmosphere at the time of the experiments was between 62·6° and 63·4° F. This detail may appear to be of secondary importance, but it cannot be neglected in an interpretation of the tracings relating to patients with reflex paralysis. Mechanical hyperexcitability and slowness of the contraction caused by percussion are particularly well marked in such cases when the external temperature is very low. These phenomena were very pronounced in two patients of this class during the winter, but became considerably less marked as the external temperature rose. It should also be noted that these patients had been undergoing treatment by diathermy. The experiments were carried out at the Collège de France in the laboratory of Prof. François-Frank, who kindly lent us the necessary apparatus.

Reading of the tracings.—The time-marking record appears on each tracing. Some of our figures show the beats of a metronome beating 200 a minute. In others, each of the main subdivisions of time marked by projections on a horizontal line represents $\frac{2}{10}$ sec. The time is measured from the percussion of the muscle, which very closely precedes the contraction. The tenths of a second which correspond approximately to the end of the contraction are accompanied by a figure; thus the number 0·3 indicates $\frac{3}{10}$ sec. from the beginning of the contraction.

In several figures the letters *a*, *b*, and *c* represent : *a* the beginning of the curve, *b* the point it which the descent becomes very feeble, and *c* the point at which the descent is definitely completed.

Interpretations of the tracings.—Our sole purpose has been

to determine the differences in the duration of the movement produced by the muscular contraction which was caused by percussion.

In the tracings the precise movement of percussion is clearly indicated; the tracing, which had hitherto been horizontal, becomes suddenly curved, and thus reveals the disturbance passively conveyed by the blow; a depression of the recording instrument is observed, sometimes preceded by a slight rise. Then after $\frac{3}{100}$ sec. the lever rises again to indicate the muscular contraction which has occurred.

We will not consider here the amplitude of the curve of contraction. It depends, it is true, on the amplitude of the movement caused by the muscular contraction, and, from this point of view, its comparative study might be of interest; but to justify any formal conclusions the intensity of the percussion would have to be measured. One would also have to be certain that no extraneous movement such as a slight displacement of the limb under examination had influenced in any way the height of the curve.

Lastly, our method of examination may possibly have modified to some extent the form of the movements. The finger, in fact, was connected with the lever of an exploring tambour. When the lever was disturbed from its position of equilibrium, it exercised on the finger an elastic traction, which in the first place limited the extent of movement during the stages of contraction, and secondly tended to restore the finger more rapidly to its state of rest during the stage of relaxation. The obvious result of this is that the total duration of the movement, as shown in our tracings, is perhaps slightly inferior to the normal. The cinematograph method alone could remedy any objection of this kind.

The end of the movement produced is marked by the return of the finger to its original position. The tracing should then resume the level which it had at first; if this did not always take place, the reason was that the fixation of the limb was not perfect enough to enable it to keep exactly its initial position.

The sign which definitely marked the end of the move-

ment produced was the return of the tracing to the horizontal. As this return was gradual, the precise moment at which it occurred could not be fixed with absolutely mathematical exactness, especially under experimental conditions such as ours. But the variations in the duration, which were inherent in the influences which we were proposing to study, proved to be so considerable that any possible error in estimation became practically negligible.

Conclusions.—We examined the reaction of muscles in three different states and under variable conditions of the external temperature. Our results were as follows— :

1. In the *normal muscle* the motor reaction varied, but was always very weak, so that the curves obtained were too ill-developed for purposes of examination; thermal influences, at least to the extent employed by us, did not cause any considerable change. Even if the curve tends to become slightly elongated as the result of the application of cold, the diminution of amplitude accompanying this change makes it almost always difficult to estimate.

2. In cases of *reflex paresis and contracture* the muscles exposed to the temperature of the atmosphere responded by a prolonged contraction; this phenomenon diminished after heat had been applied, but considerably increased after a previous chilling of the hand examined.[1]

[1] In this connection we will remind the reader that the influence of cold on the form and duration of the muscular contraction caused by electrical stimulation has been established for a very long time. An account of this question will be found in the very interesting article of Maurice Mendelssohn (229) on the study of electrical diagnosis. Grund has shown that the essential characters of the partial reaction of degeneration (slowness of the contraction and predominance of the anode over the cathode) can be produced *normally* by cold and then be caused to disappear by heat.

With this we may compare the slowness of the contraction caused by mechanical stimulation, observed by us in *patients suffering from reflex paresis or contracture.* But it should be mentioned that in the patients in question *the artificial application of cold is only a means of making evident the abnormal state of the muscular contractility in ill-developed cases.* A slight chilling of short duration without any apparent action on the muscles of the sound side is enough to render evident the mechanical hyperexcitability of the

3. In muscles presenting the *reaction of degeneration*, at the temperature of the atmosphere, the contraction has been as long as in the previous cases, and after the action of heat the slowness became less marked. But after the application of cold, the amplitude of the contraction became too weak for it to be analysed.

4. One last word in conclusion. The term " atmospheric temperature " obviously lacks precision. In our experiments we had a moderate summer temperature. A low external temperature would clearly supply our artificial conditions for chilling.

After this description of the circumstances connected with our experiments we will reproduce some of the curves, and give a short account of them.

FIRST SERIES OF EXPERIMENTS

(*Tracings I–V*)

Records of the movements of the thumb and little finger caused by percussion of the thenar and hypothenar eminences, at the atmospheric temperature, after application of heat or cold. The tracings of the contractures of the thumb muscles only are shown, those of the little finger being exactly alike.

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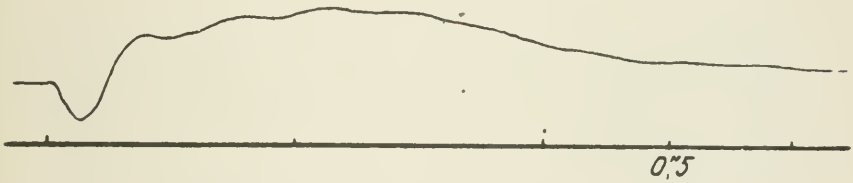
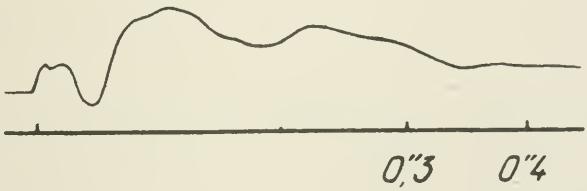
muscles and to make the muscular contraction on the affected side considerably slower. Besides, in definite cases of reflex nervous disorders, mechanical hyperexcitability is usually present. As regards the slowness of the muscular contraction, it appears spontaneously in the affected limb in winter, and sometimes even in hot weather, when the two sides are under identical conditions and without it being necessary to have recourse to the artificial application of cold. Mechanical hyperexcitability of muscle and slowness of the contraction are therefore associated, to a considerable extent at least, with the natural hypothermia which appears to result from reflex vaso-constriction following the lesion. We are dealing therefore not with a physiological but with a pathological reaction.

A. Reflex Contractures and Pareses

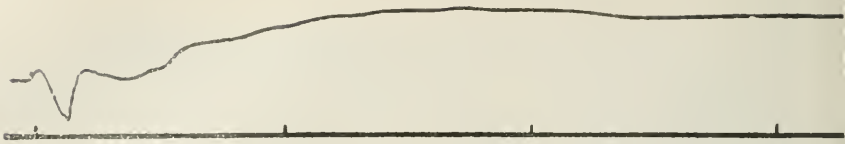
Case I.—M. . . . Contracture of the hand in the form of a fist (*v.* Pl. VI, p. 564) following perforation of the forearm (December 1914) with disturbance of electrical reactions. Intense hypothermia and vaso-motor disorders with considerable diminution of the amplitude of the oscillations (*v.* Table I, Case II, p. 668). The mechanical hyperexcitability of the muscles of the hand is always present, but the slowness of the contraction is most evident when the external temperature is cold. At the time of the experiment (June 1916) the contraction on the affected side lasted 0·6 sec. at the temperature of the atmosphere (18° C.), 0·4 sec. after application of heat, and 1·4 sec. after application of cold (Tracings I, II, and III).



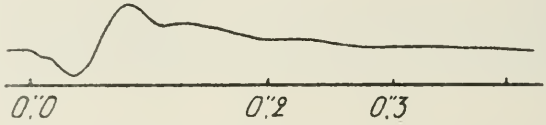
Tracing I (Reflex Contracture, Case I).—*At the atmospheric temperature* (18° C.) the contraction lasts from 0·5 sec.–0·6 sec. on the affected side (and 0·4 sec. on the sound side). The local temperature of the thenar eminence is 15° C. (and 23° C. on the sound side). The maximum oscillations (Pachon) measure 0·25–0·50 division (and 3 divisions on the normal side).



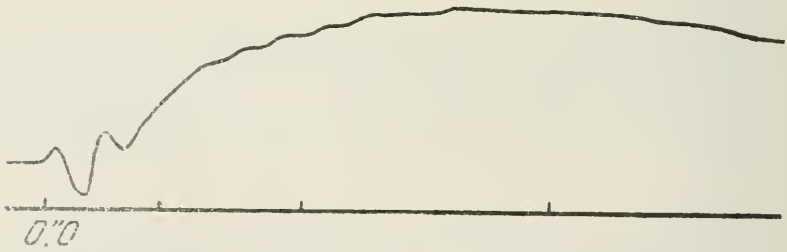
Tracing II (Reflex Contracture, Case I).—*After application of heat* (*of short duration*) by immersion of the two hands in water at 44° C. the contraction lasts from 0·3 sec.–0·4 sec. on the affected side (and 0·3 sec. on the sound side). The local temperature of the thenar eminence is 28° C. (and 27° C. on the sound side).



Tracing III (Reflex Contracture, Case I).—*After application of cold (of short duration)* by immersion in water at 12° C. the contraction lasts 1·4 sec. (it is difficult to measure the exact duration of the contraction on the sound side, in which the amplitude diminishes considerably under the action of cold; it appears to last from 0·2–0·3 sec. only. The local temperature of the thenar eminence in the wounded limb is 12° C. (and 14° C. in the normal limb).

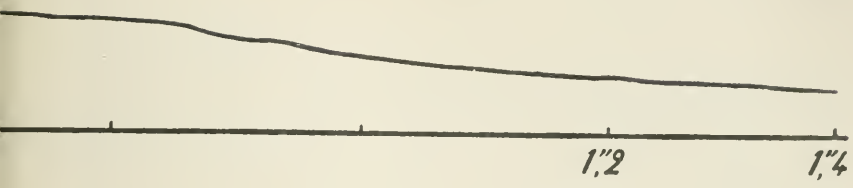


Tracing IV (Reflex Paresis, Case II).—*At the atmospheric temperature* (18° C.) the contraction lasts 0·2 sec.–0·3 sec. on the affected side (and 0·2 sec. on the sound side). The local temperature of the thenar eminence is 27° C. (and 25° C. on the sound side). The maximum oscillations (Pachon) are equal on both sides and measure 3 divisions.



Tracing V (Reflex Paresis, Case II).—*After application of cold (of short duration)* by immersion in water at 13° C. the contraction lasts 1·2 sec. on the affected side (and 0·5–0·6 sec. on the sound side). The local temperature of the thenar eminence is 14° C. (and 15° C. on the sound side). The amplitude of the oscillations is *nil* on the affected side (and 1 division on the sound side).

Case II.—T. . . . Paresis of the hand and fingers following a wound of the posterior surface of the forearm without obvious disturbance of the electrical reactions. Local

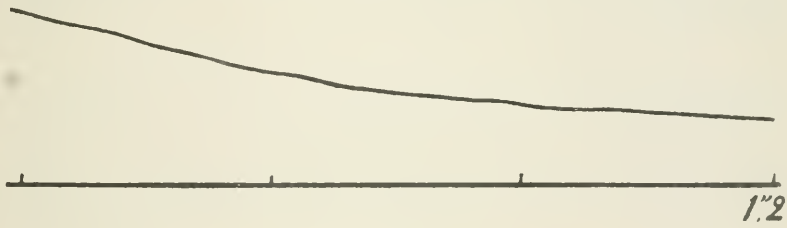


hypothermia and marked and obvious vaso-motor phenomena. Mechanical hyperexcitability of the muscles of the hand and slowness of the contraction very marked during the winter season. At the time of the experiment (June 1916) the contraction of the affected side lasted 0·2 sec.–0·3 sec. at the temperature of the atmosphere (18° C.), and 1·2 sec. after a short application of cold (Tracings IV and V).

B. Paralyses with Reaction of Degeneration

(Summary of results without Tracings)

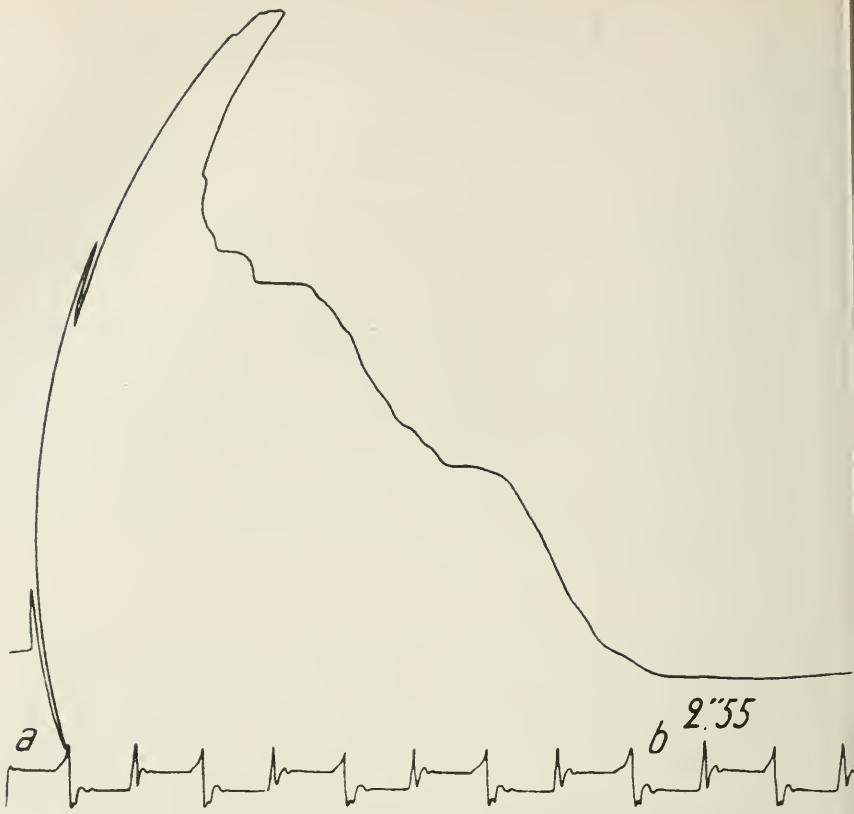
Case III.—D. . . . Paralysis of ulnar and median with complete R.D. of the muscles of the thenar and hypothenar eminences (record of movements of thumb).



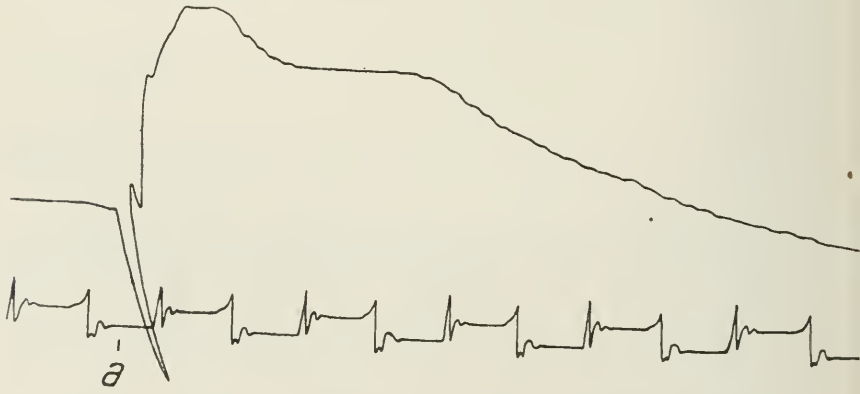
At the atmospheric temperature 18° C., the local temperature of the thenar eminence being 18° C., the duration of the contraction was 1·2 sec.

After application of heat, the local temperature of the thenar eminence being 27° C., the duration of the contraction was 0·6 sec.

Case IV.—B. . . . Paralysis of ulnar nerve with marked



Tracing VI (Reflex Contracture, Case I).—*At the atmospheric temperature* (18° C.) *the contraction lasts* 2·55 *sec.* on the affected side. Local temperature: 14° C.



Tracing VII (Reflex Contracture, Case I).—*After application of cold* (*of short duration*) by immersion in water at 12° C. the contraction lasts 5·1 sec. on the affected side. Local temperature: 9° C.

R.D. of the hypothenar eminence (record of movements of little finger).

With an atmospheric temperature of 18° C. and a local temperature of 18° C. the duration of the contraction was from 1·2 to 1·3 sec.

After application of heat the local temperature being 27° C., the duration of the contraction was from 0·6 to 0·8 sec.

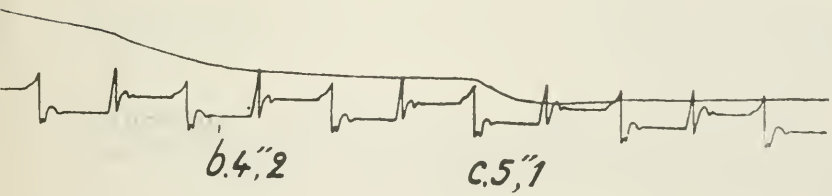
SECOND SERIES OF EXPERIMENTS

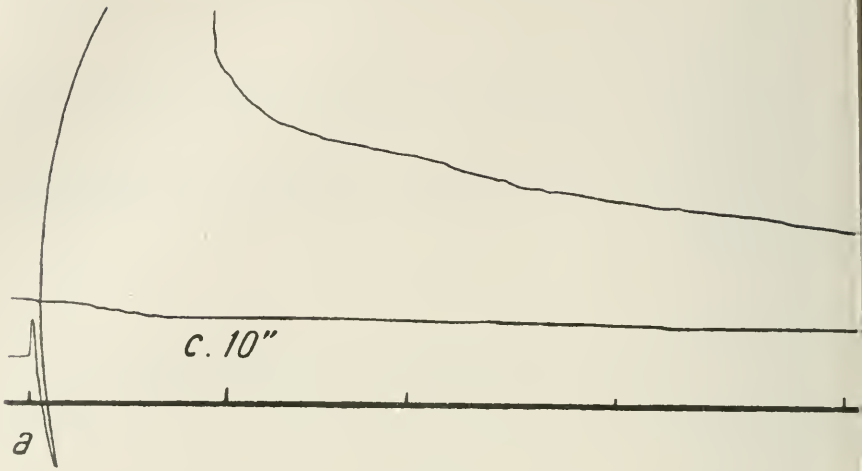
(*Tracings VI–X*)

The speed of the cylinder was reduced and the length of the recording lever increased to make the descending phases of the curve more pronounced and to facilitate a more accurate measurement of the duration of the prolonged contractions. (The terminal part of the curve which ended very gradually was not shown in the tracings of the first series of experiments.)

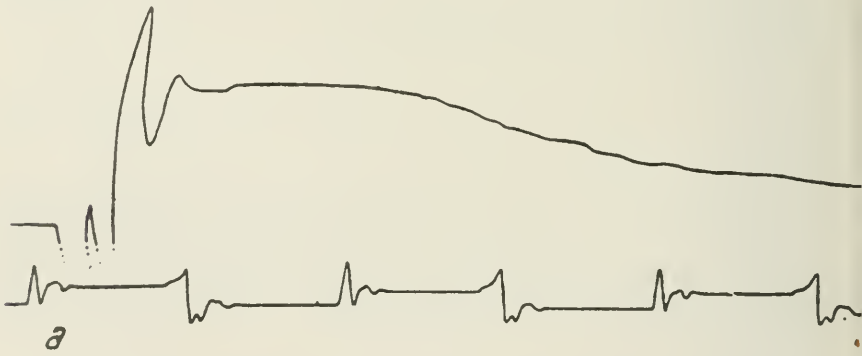
A. Reflex Contractures and Paralyses

Case I (*v.* p. 548).—On the affected side the contraction lasted 2·55 sec. at the atmospheric temperature (18° C.), and 5·1 sec. after application of cold (Tracings VI and VII). On the sound side the contraction was too faint to be recorded under the circumstances.





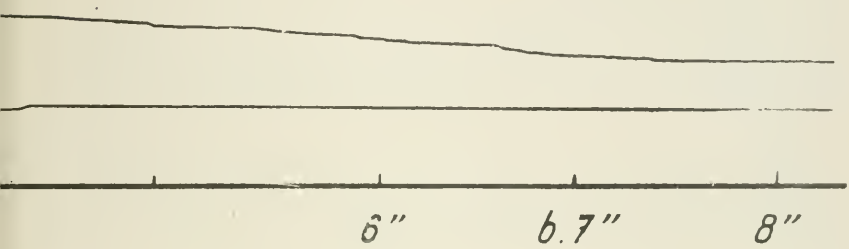
Tracing VIII (Reflex Paresis, Case II).—*After application of cold* (*of short duration*) to the two hands in water at 12° C. the contraction on the affected side lasts 10 sec., the temperature of the thenar eminence being 12° C. On the sound side the contraction after application of cold is of too weak amplitude to be recorded.



Tracing IX (Paralysis with Reaction of Degeneration, Case III). —*At the atmospheric temperature* (17·5° C.) the contraction lasts 3·15 sec. on the affected side, the local temperature of the thenar eminence being 23° C.

Case II (*v.* p. 546).—At the atmospheric temperature (18° C.), the contraction on the affected side lasted 1·8 sec., the local temperature being 12° C.

After application of cold (Tracing VIII) the contraction lasted 10 sec. Note that this tracing is on a different scale from the others. Each of its divisions corresponds to 1 sec., whereas in the other tracings of the same series of experiments it corresponds to 0·3 sec., and in the tracings of the first series to 0·2 sec.

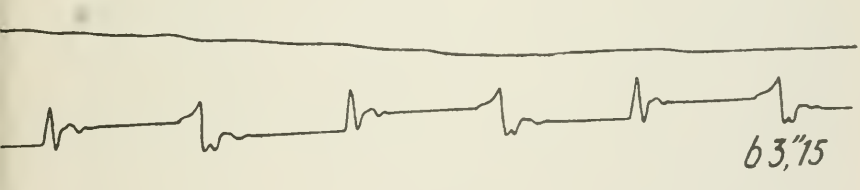


B. Paralyses with Reaction of Degeneration

(*Tracings IX and X*)

Case III.—B. . . . Paralysis of ulnar nerve with marked R.D. of hypothenar eminence (record of movements of little finger).

At the atmospheric temperature the contraction on the affected side lasted 3·15 sec. (Tracing IX).



After application of heat the contraction lasted 2·4–3 sec. (Tracing X).

After a short application of cold by immersion in water at 12° C., the extent of the contraction on the affected side was too feeble to be recorded.

On the sound side the amplitude of the contraction was also too feeble to be recorded.

Tracing X (Paralysis with Reaction of Degeneration, Case III). —*After application of heat* by immersion of the two hands in water at 44° C., the contraction lasts 2·4 sec. on the affected side.

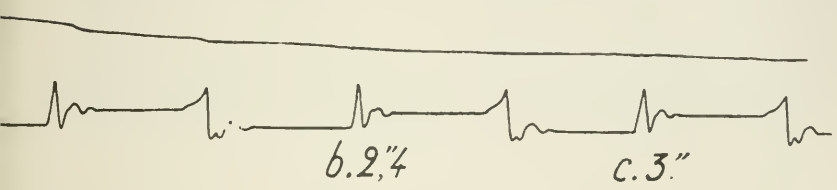
The graphic method, therefore, confirms the result of numerous clinical investigations; it shows that the slowness of the muscular contraction caused by percussion may be as marked in reflex contractures and pareses as in many cases of paralysis with reaction of degeneration. The intensity of the muscular hyperexcitability and slowness of the contraction are in proportion to the vaso-motor and thermal disorders; when there is considerable hypothermia, it may almost be asserted a priori that the mechanical excitability of the muscle is exaggerated and that the muscular contraction is slow.

But what relation is there between vaso-motor and thermal disorders, on the one hand, and disturbances of voluntary movement on the other? There is undoubtedly a connection between the two classes of phenomena; characteristic vaso-motor disorders do not necessarily involve, it is true, a profound disturbance of the motor functions, but they are accompanied by some impairment of energy. We will add

that some reflex motor disorders are not accompanied by any vaso-motor or thermal disorders.

Electrical and Mechanical Excitability of Nerves

Mechanical hyperexcitability of muscles has often corresponding to it some quantitative modifications in electrical contractility, such as *faradic and voltaic hyperexcitability of muscles,* or, on the contrary, slight



hypoexcitability and sometimes *premature fusion of contractions. There is never any reaction of degeneration.*

The following is a description of premature fusion of contractions which is easy to demonstrate when it is unilateral, and is more readily detected by touch than by sight. When one applies to the muscle the two electrodes of a volta-faradic apparatus and holds the handles between the fingers, if the number of interruptions is progressively increased, at a certain moment the vibrations corresponding to the contractions cease completely on the affected side, while on the sound side with the same number of interruptions the vibrations still continue to be felt. This occurrence is the consequence of the slowness of the muscular contractions on the affected side (182).

We may add that *mechanical hyperexcitability of the nerve trunks* may be occasionally observed. Percussion of the ulnar nerve in the space between the olecranon and internal condyle causes a characteristic movement of adduction of the thumb with flexion of the first phalanx (adductor pollicis), combined with a movement of flexion of the fourth and fifth fingers

at the first phalanx (interossei). Percussion of the posterior tibial nerve causes a movement of flexion of the toes.

Mechanical hyperexcitability of the nerves appears like muscular hyperexcitability to be closely associated with vaso-motor disorders and hypothermia; the application of heat may temporarily reduce or abolish it.

Muscular Hypotonus

Hypotonus is sometimes very pronounced and may be as great as in the most serious nervous lesions, but its area is usually restricted. It may be very marked in certain muscular groups of the affected limb, and be completely absent in others. Thus it may be

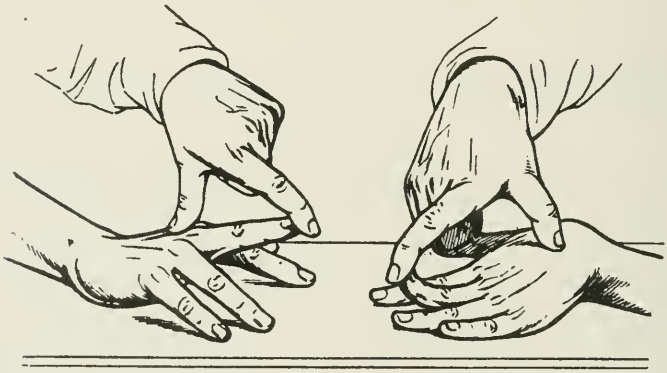


Fig. 8.—Hypotonus of the flexors of the fingers in a case of contracture of the flexors of the hand with hyperextension of the fingers (*v.* Pl. VII, p 572).

possible to obtain by passive movements in various cases a hyperflexion of the hand forming a very acute angle with the forearm (*v.* Figs. 4 and 10), hyperextension of the fingers in an arc of a circle (*v.* Figs. 8 and 9), hyperflexion of the thigh (leg extended) far beyond a right angle (*v.* Fig. 12), and hyperflexion of the leg combined with hyperflexion of the thigh, a complex movement in which the thigh is brought close to the abdominal wall and the heel to the gluteal region

(*v.* Figs. 13 and 15). Owing, however, to individual variations in tonicity, no attention should be paid to hypotonus unless it is definitely asymmetrical.

When it is very well developed, it may even be

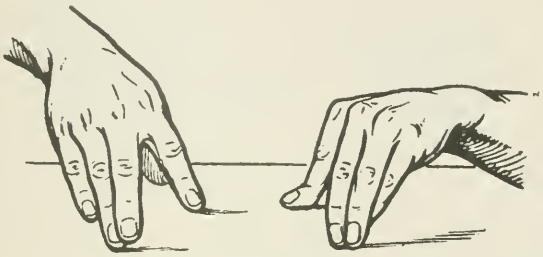


FIG. 9.—HYPOTONUS OF THE FLEXORS OF THE FINGERS in a case of accoucheur's hand (*v.* Pl. V, p. 526).

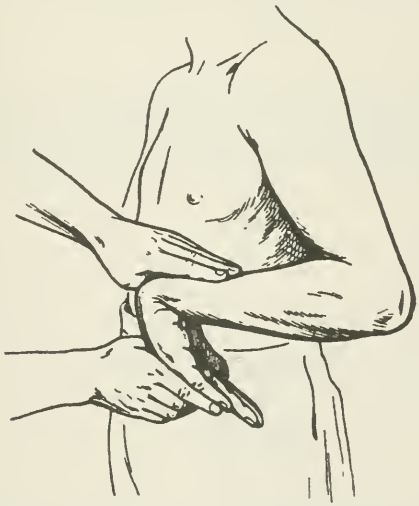


FIG. 10.—HYPOTONUS OF THE EXTENSORS associated with contracture of the flexors of the hand (*v.* Pl. IV, p. 524).

seen in the patient's movements. If he raises his arm suddenly or is walking, the affected hand swings passively in every direction as if it did not belong to him and was simply controlled by the laws of physics.

Lastly, when hypotonus of certain muscle groups

is associated with contracture of their antagonists, which is a frequent occurrence, it may give rise to actual subluxations.

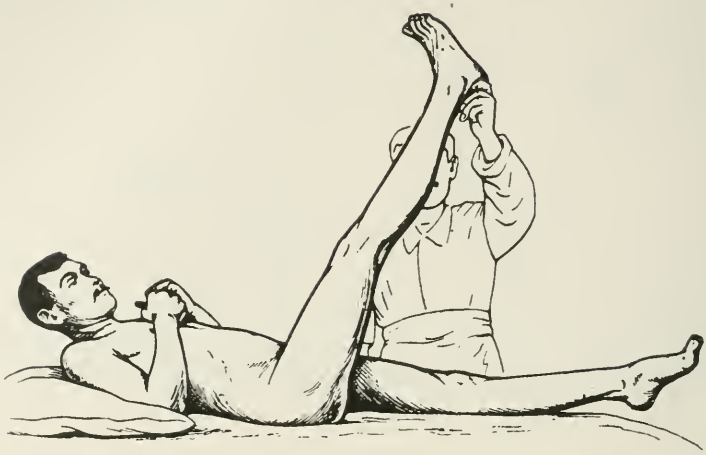


Fig. 11.

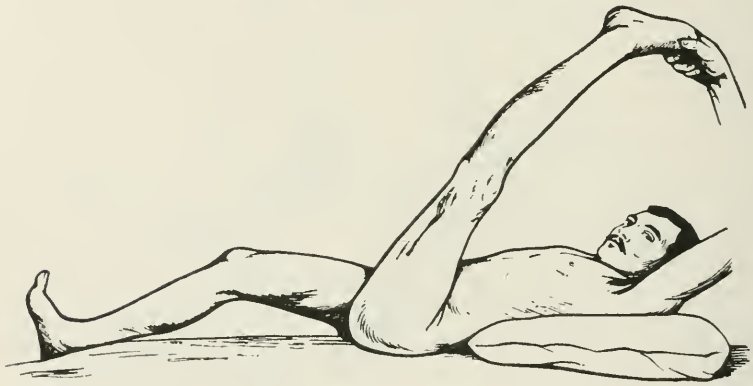


Fig. 12.

FIGS. 11 and 12.—HYPOTONUS OF THE EXTENSORS OF THE HIPS ON THE PELVIS in a case of contracture of the foot and quadriceps with fibro-tendinous retractions after a wound of the thigh by a shell fragment (September 1914), followed by prolonged suppuration. Marked vaso-motor symptoms. Exaggeration of the ankle jerk (July 1916). Persistence of the contracture and exaggeration of the tendon reflexes in the affected foot during chloroform narcosis (*v.* Appendix, Case XIII, p. 666). Compare the amplitude of the passive movement produced on the affected side (Fig. 12) with that on the sound side (Fig. 11).

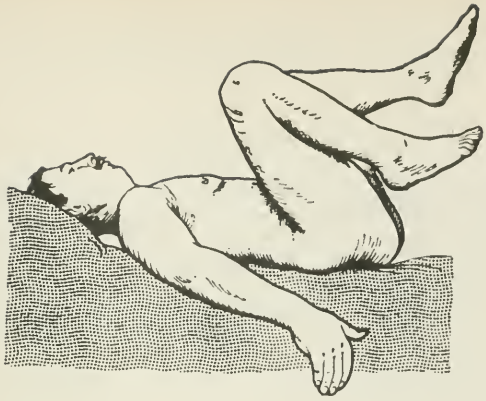


Fig. 13.

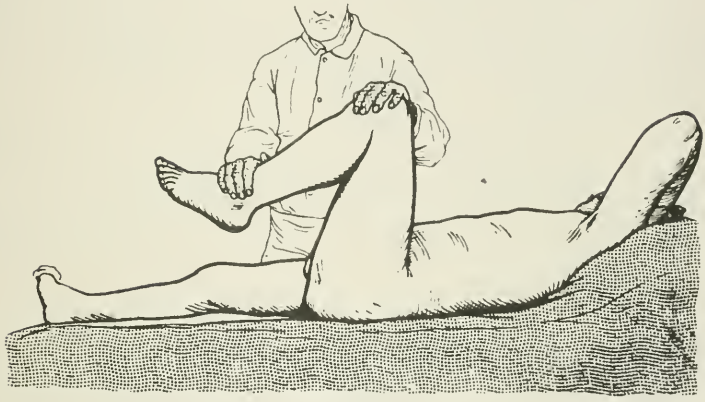


Fig. 14.

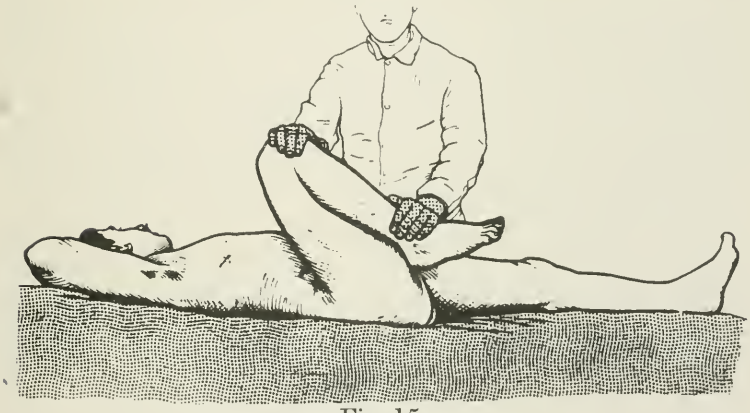


Fig. 15.

FIGS. 13, 14 and 15.—HYPOTONUS OF THE RIGHT QUADRICEPS in a case of reflex paresis of the muscles of the leg and foot following a shrapnel wound of the thigh (September 1914). Marked vaso-motor symptoms and hypothermia. Mechanical hyper-excitability of the muscles of the foot and leg without obvious disturbance of the electrical reactions (September 1916).

Amyotrophy

We have already stated that amyotrophy sometimes constitutes the most prominent feature of the case. Thus traumatism or arthritis affecting the knee may be followed by very pronounced atrophy without the paresis being very pronounced. On the other hand, paresis and contracture may be well developed and the amyotrophy be only of secondary importance.

The following figures show the degree of diminution in the size of the muscles. The circumference of the affected leg is 3 to 6 centimetres smaller than that of the sound leg.

In the thigh the difference is frequently 3 to 5 centimetres and sometimes 7 or 8 centimetres.

In the upper part of the forearm it may be 2 to 3 centimetres, and in the upper arm 3 to 4 centimetres.

Exaggeration of the Tendon Reflexes

Exaggeration of the tendon reflexes, when it undoubtedly exists, is a sign of great value. It may be difficult to determine, for the reflexes on the sound side are sometimes rather brisk. The only criterion is a definite asymmetry between the two sides. We may remind the reader in this connection that in judging of the value of a tendon reflex it is not merely the amplitude of the movement, but its others characters as well that must be taken into account, such as suddenness and polykinesis (179).

The tendon reflexes appear to be more frequently exaggerated in contractures of the lower limb than in the hypertonic forms observed in the upper limb. This difference resembles that which we have already noted in connection with muscular rigidity. Nevertheless, even in reflex contractures of the lower limb, exaggeration of the tendon reflexes is often open to doubt, and may be entirely absent. Sometimes it is latent only, and can then be made evident by chloroform narcosis (*v.* pp. 563 and 632)

Loss of Cutaneous Reflexes

In the cases which we have observed sensory disorders were generally accompanied by loss of the plantar cutaneous reflex. After immersion in hot water the cutaneous reflex reappeared for a time, and was then identical with the reflex on the sound side. This experiment has been performed several times in numerous patients, and the same result has always been obtained.

These disorders, therefore, should be grouped with the remarkable modifications seen under the influence of heat in the mechanical excitability of nerves and muscles, and the diminution in amplitude of the oscillations.

This is therefore a characteristic peculiar to reflex nervous disorders (145).

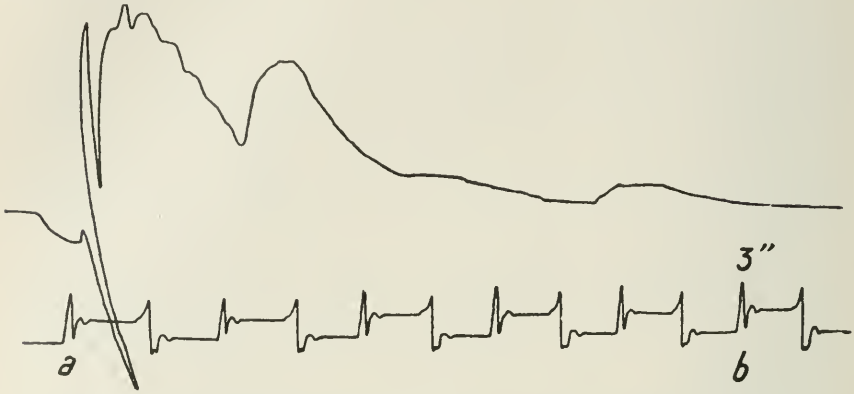
There is every reason for supposing that the loss of the cutaneous reflexes is due to a circulatory disturbance, and in this connection we will quote some remarks made by one of us relating to changes in the cutaneous reflexes after compression by Esmarch's band—

"It is well known that compression of a limb by Esmarch's band may cause a loss of the tendon reflexes in that limb, even in pathological cases in which there is an exaggeration of the tendon reflexes and ankle clonus. From experiments made on patients suffering from paralysis with contracture due to lesions of the pyramidal system and presenting an extensor response, I found that under the influence of a similar constriction applied to the lower limb changes were also occasionally present in the cutaneous reflexes. When the compression has been sufficiently prolonged, the toes no longer respond to stimulation of the sole, but remain motionless" (178).

Tremor

The affected limb is frequently subject to tremor (P. Marie and Foix); we found that this symptom was most pronounced during the hot weather, when the

limb had been artificially warmed, or when the patient exerted himself.



Tracing XI (Reflex Contracture, *v.* Case I, p. 549). Undulations on the tracing of the movement of the thumb caused by percussion of the muscles of the thenar eminence at the atmospheric temperature. The undulations are more marked still after application of heat.

In the course of our investigations with the graphic method we noted on the tracings certain waves corresponding to the mechanical muscular contraction. After application of heat the existence of very definite secondary muscular contractions was also found for a time.

These tracings, it may be said in passing, appear to have some resemblance to those illustrating muscles under the action of veratrine as described by Mlle. Joteyko in her book (212).

Sensory Disturbances

Patients with reflex contractures occasionally complain of painful sensations; the pains are either spontaneous or caused by pressure on the nerve trunks. Vulpian drew attention to them long ago, and they have been mentioned again by Gougerot and Charpentier (136), Guillain and Barré (138), and Tinel (154).

In several cases of reflex paralysis or paresis of the

lower limb, we have observed hypæsthesia with a more or less segmentary distribution. It was most marked on the sole, less developed on the dorsal aspect of the foot, and least of all on the lower two-thirds of the leg. These sensory changes were present with all forms of stimulation, including touch, pain and heat. Disturbance in the sense of position was also noted. The sensory disorders sometimes diminished after immersion of the part in hot water.

In some cases of acromyotonus Sicard (157) observed deep anæsthesia of the hand and forearm. He was struck by the absence of any reaction to violent stimuli applied to the affected areas; faradisation by a coil with fine wire, injection of 95 per cent. alcohol into the subcutaneous tissue or muscles, and even an operation without an anæsthetic, local or general, did not cause any sign of pain, any vaso-motor change in the face or acceleration of the pulse. "What are we to conclude from these cases?" writes Sicard. "Either we must suppose that these patients are determined not to betray themselves in the course of these tests, and possess an extraordinary will power, though for my part I refuse to believe in the existence of so much moral energy in the face of such physical pain; or we must consider that this is an example of hysterical phenomena, and that hysteria alone is capable of producing a *real* superficial and deep anæsthesia, though this does not agree with the teaching of Dr. Babinski, and with our own experience at La Salpêtrière. Our hysterical patients in the 'Clinique Charcot,' with a typical hemi-anæsthesia or an anæsthesia of a segmentary type, could never undergo an operation with the knife on their insensitive limb without a local or general anæsthetic.

"Or, lastly, the hypothesis must be accepted—and we concur in this view—that in cases which have undergone such tests, the anæsthesia is real, independent of the patient's will, and does not obey the rules of pithiatism. Such phenomena must be regarded as possessing a real autonomy, and may be explained by

the reflex pathogeny suggested by Drs. Babinski and Froment."

Secretory and Trophic Disorders

Disorders of secretion.—It is not uncommon to find that the skin is damp, especially at the extremity of the affected limb; sometimes it is even slightly macerated (*v.* Pl. VI, p. 564).

We saw a very pronounced example of hypersecretion of sweat in a captain who presented characteristic reflex disorders after perforation of the lower part of the forearm. This officer also told us that the secretory disorders which appeared on the affected side shortly after the wound involved the sound hand four months later. We several times had the opportunity of seeing beads of sweat appear on both his hands under the influence of various causes (164)

Trophic disorders.—A general atrophy may be found, especially when the lesion is situated on the hand, and this can be clearly seen in the tapering fingers. A comparison of the skiagrams of the two hands shows *decalcification of the skeleton*, which has been observed after traumatism of the limbs by a large number of writers. It is best seen in the extremities. In the X-ray plate the skeleton of the affected hand appears to be more distinct and the outline of the bony trabeculæ clearer and more delicate, while the epiphyses and joint surfaces are less distinct than on the sound side. The same appearances are to be found, but in a less degree, in the bones of the forearm.

There is almost always a certain degree of *fibro-tendinous* or *muscular retractions* According to the position of the contracture these may be seen in the joints of the fingers, wrist, elbow, hip, foot and ankle, but they are most marked in the finger-joints, which are frequently enlarged and painful like those of chronic rheumatism. They develop rapidly, and explain to a certain extent the limitation and difficulty in the movements of the joints. Although they are hardly

ever completely absent in reflex contractures and pareses, their intensity varies. On the other hand, they are exceptional in cases of hysterical or pithiatic paralysis or contracture. If the contrary has sometimes been asserted, this is doubtless due to the frequent confusions which have hitherto been made, or to the failure to recognise the existence of hystero-organic or hystero-reflex associations.

It may be noted that *hypertrichosis*, which formed the subject of an interesting study by Villaret may be observed not only in neuritis but also in reflex contractures and pareses. We may remind our readers that it is absent in purely hysterical contractures and paralyses.

As a rule the nails also show some changes in patients with reflex disorders, their growth being less rapid than on the sound side.

Examination during Chloroform Narcosis

We have already mentioned the characteristic signs which were revealed by examination of reflex contractures under chloroform, viz. *exaggeration of the tendon reflexes* in the affected limb, *spasm* caused by any attempt to correct the vicious attitude, and *persistence of the contracture to an advanced stage of chloroform narcosis* (*v.* p.503). The summary of our principal observations relating to this question will be found below (*v.* Appendix, pp. 659 *et seq.*); we will merely state here that all these cases except one were examples of contracture of the lower limb. The exception was an instance of an "accoucheur's hand," in which the contracture did not persist until an advanced stage of the narcosis, but disappeared at the beginning. But the ulnar pronator reflex, which was almost normal during the waking state, was definitely exaggerated on the affected side.

We have already described to the best of our ability the way in which the reflex nervous disorders which we have studied appear and become established. It is impossible to give an exact account of their further

Plate VI

A. Contracture of the extensors of the hand and fingers with hypotonus of the flexors following a wound of the forearm complicated by fracture of the radius. Vaso-motor symptoms with œdema of the hand. Hyperidrosis and macerated appearance of the skin. Marked trophic troubles (general atrophy of the fingers, decalcification of the skeleton, trophic changes of the nails, and amyotrophy), very marked mechanical hyperexcitability of the muscles of the hand, radial muscles and extensors of the fingers, and also of the ulnar nerve (January 1916).

B. Contracture of the flexors of the fingers with overriding of the fingers following a perforation of the forearm by a bullet (December 1914), which appeared immediately after the wound and persisted unmodified since (February–October 1916). Very marked and obstinate vaso-motor phenomena (diminution of the amplitude of the oscillations) and thermal disorders. Faradic, voltaic and especially mechanical hyperexcitability of the muscles of the hand and forearm. Very pronounced slowness of the contraction during the winter or after a short application of moderate cold, which had no effect on the sound side (*v.* Tracings III and VII, pp. 546 and 548). Tremors and muscular twitchings during hot weather.

C. Accoucheur's hand with extensive contracture of the forearm following a wound of the arm (August 1915), complicated by diffuse cellulitis. Vaso-motor symptoms and hypothermia. Hand mottled and succulent. Mechanical hyperexcitability of muscles. Exaggeration of ulnar pronator reflex on the affected side with unilateral clonus during chloroform narcosis (*v.* Appendix, Case X, p. 665).

PLATE VI

development, as the cases under our observation are of too recent a date.

All that we can say with certainty is that these disorders when left to themselves may remain stationary for a very long time without undergoing any appreciable modification, just as if they were permanent. It should be mentioned in this connection that Charcot and Vulpian were impressed long ago by the obstinate character of reflex amyotrophies caused by slight lesions which had long since disappeared.

We have often observed a progressive improvement in contracture and paresis as a result of the various modes of treatment which we shall describe later. But putting aside the hysterical element which may be associated with them to a greater or less extent, reflex disorders are slow in showing any change. In the most favourable cases, including those in which there was apparent recovery, we have always seen persisting hitherto a state of weakness characterised by reduction of muscular power and the impossibility of prolonged physical effort. This is a point which must be considered by medical boards. There is no reason for supposing that a return to normal cannot be finally obtained.

CHAPTER VIII

PATHOGENY OF THE SO-CALLED REFLEX NERVOUS DISORDERS

Vulpian and Charcot endeavoured to establish the pathogeny of these disorders, which even then appeared to them to form a well-defined group of nervous manifestations. After rejecting the hypothesis of a lesion of the nerve centres they discussed the question as to whether these phenomena, and especially the amyotrophy which had particularly impressed them, could not be attributed merely to immobilisation. "In dealing with the mechanism," writes Vulpian, "of muscular atrophy associated with lesions of the joints, John Hunter had propounded an hypothesis which has merely an historical interest (effect of sympathy) . . . but the celebrated English surgeon rejects the opinion previously held, which attributed the atrophy to inertia of the limb, by observing that in cases of unilateral arthritis the muscles of the sound leg do not atrophy, although they are also at rest. This objection is of little value, since it is based on an observation which is not strictly accurate. It is well known that in arthritis of the knee, for instance, though the corresponding limb keeps almost motionless, the other limb carries out more or less extensive and repeated movements.

"But a more powerful objection could have been derived from the fact that the limb affected by arthritis is not always condemned to absolute rest. Even the muscles undergoing atrophy sometimes carry out more or less extensive movements."

Charcot has several times insisted on the impossibility

of explaining amyotrophy of articular origin by immobilisation. In his treatment of the subject in his Tuesday lectures (1888–9) he makes the following remarks: "This was obviously a case of paralysis, and the amyotrophy which followed as a result of the articular affection appeared very rapidly, since in six weeks' time it was considerable. Under such circumstances it is obviously not worth while discussing the theory which would attribute amyotrophy of this kind to the influence of prolonged muscular inactivity, the complete duration of which was not more than four or five days. Nor can it be admitted that these amyotrophies are the result of a propagation of an inflammatory process to the muscles, for the arthritis in our case was very slight; besides, it would be very difficult to explain so rapid an extension of an inflammatory process throughout the limb with a marked predominance in the anterior muscles of the thigh.

"I have long since adopted and maintained Vulpian's theory, which appears to me to be the only one suitable for the explanation of all the cases which I have just described. This theory, as you know, is that the centripetal articular nerves which have been irritated at their peripheral extremity by the affection of the joint transmit the irritation to the spinal grey substance, or more precisely to the large nerve cells in the anterior cornua, with the effect that amyotrophy results. A German doctor recently taxed me with 'mysticism' for supporting this theory. I must say that I am puzzled by such an accusation, for I fail to see what mysticism there can be in a theory founded on anatomical and physiological analogies.

"The pathogeny according to which amyotrophy in these cases is the result of inaction is not the only one which has been opposed to the reflex pathogeny, for some authors have suggested the hypothesis of an ascending neuritis. As the result of experimental researches by Hayem (117), who had produced on some animals changes in the cells of the anterior cornua

by wounds of the nerves, Vulpian himself had at first wondered if these nervous disorders might not be due to similar lesions. But he showed that one could not hastily apply all the results of experimental work to clinical medicine, and also that this ascending neuritis never occurred in some animal species (the dog, in particular), although it was possible to reproduce in them all the phenomena in question."

" In almost all the cases," he added, " for which we are seeking an explanation we are not dealing with direct and more or less violent lesions of the nerve trunks, but with irritation affecting the nerve filaments or even the extremities of the nerves (in arthritis, for example); so that the conditions are quite different from those under which Dr. Hayem made the experiments which he has published. Lastly, it must not be forgotten that Dr. Valtat examined the nerves in animals which showed muscular atrophy after experimental arthritis and found them perfectly normal. Therefore, until there is a direct proof of the existence of an ascending neuritis in man, in cases of muscular atrophy produced by peripheral lesions (arthritis, wounds, etc.), I think we may continue to attribute this atrophy to a change in the grey subtance of the spinal cord following a special irritation arising at the site of the lesion, and acting on the spinal centre by the agency of the centripetal nerves.

" Although I dispute the hypothesis of ascending neuritis as a general theory for the so-called reflex muscular atrophy, I do not hesitate to admit the real existence of this neuritis and its consequences in certain special cases."

Thus we find that Charcot and Vulpian finally support the theory of nervous disorders of reflex origin. But what is the exact meaning to be given to this term? " A musculo-motor reflex action," says Vulpian, " necessarily requires the co-operation of three factors at least (apart from the starting-point of the stimulus and the effect produced), viz. (1) A centripetal nerve which conveys the stimulus to a nerve centre. (2) A

nerve centre which receives this stimulus and transforms it into a motor impulse. (3) A centrifugal nerve which conveys this motor impulse to the periphery."

Are these three factors to be found in cases of so-called reflex muscular atrophy? The impulse produced by the peripheral lesion is certainly transmitted to the grey substance, and appears to affect it to a certain extent. But Vulpian, who was considering simple amyotrophy and amyotrophic paralysis only, and regarded exaggeration of the tendon reflexes as exceptional, considered that he had a right to assert that the centrifugal impulse was absent. We should thus be dealing with a sort of inhibitory action due to the peripheral stimulus and affecting the motor and trophic functions or the trophic function exclusively.

Charcot showed that not only amyotrophy and paresis but also contracture and exaggeration of the tendon reflexes, which he regarded as the rule, bore a close resemblance to a reflex action. "Affections of the joints," he writes, "when they involve the spinal centre, sometimes excite the nerve cells which give rise to muscular contracture, or, on the contrary, depress them and so produce amyotrophic paralysis. . . . It should be added that these two varieties of disturbance of the spinal centres may be combined in the same subject." "Even in cases of amyotrophic paralysis," adds Charcot, "to judge at least by the cases which I have shown you, the preliminary stage of muscular contracture, viz. exaggeration of the reflexes, is to a certain extent combined with the amyotrophy. . . . These two groups of phenomena represent, so to speak, the extreme poles of the same morbid process."

Exaggeration of the tendon reflexes and contracture testify to the existence of the centrifugal impulse which Vulpian required to identify the so-called reflex disorders with reflex phenomena properly so called.

However that may be, both in cases of flaccid paralysis with amyotrophy and of contracture with exaggeration of the tendon reflexes, the existence of a disturbance of the spinal centres following a peripheral

irritation was admitted unhesitatingly by Charcot and Vulpian.

The results of our researches confirm the views of these writers and furnish new arguments in support of their theory.

Exaggeration of the tendon reflexes which Charcot had shown to be so frequent in these cases is even more common than might be supposed, since *examination during narcosis* (*v.* pp. 563 and 632) reveals it in some cases in which it is latent in the waking state.

Sometimes, even when there is an absence of any exaggeration of the reflexes, stimulation of the spinal centres and the centrifugal impulse are revealed by the spasms produced during chloroform narcosis.

But can we explain by a reflex action all the phenomena which form part of the syndrome under discussion and especially the vaso-motor and thermal disorders? This is a question which we shall now proceed to discuss.

We have long been impressed by the importance of the vaso-motor and thermal disorders of which we have endeavoured to give an accurate clinical description, and which indicate a disturbance of the *sympathetic* system. In an interesting study of the pathology of the sympathetic system, H. Meige and Mme. A. Benisty emphasised the part which it seemed to play in the production of the nervous manifestations with which we are concerned.

What is the nature of this disorder? Is it a paralysis? This view does not seem to agree with the facts observed. Ever since Claude Bernard's memorable experiment it has been known that section of the cervical sympathetic is followed by vaso-dilatation with rises of the local temperature and intra-arterial pressure. There is every reason to believe that a destructive lesion of the sympathetic tracts in the limbs may cause phenomena of the same kind.

We may note in this connection that laceration of the sympathetic plexus surrounding the brachial artery which has been carried out for therapeutic purposes in

patients suffering from causalgia (Leriche's operation), has been followed by pronounced vaso-dilatation with increase of 2–4 cm. in the arterial pressure and rise of the skin temperature. These phenomena which appeared one hour after the recovery of the patient from the anæsthetic were still perceptible a week later (Heitz).

Now, in the cases with which we are concerned, we are dealing with vaso-constriction, as is proved by the result of our oscillometric observations. There is, therefore, a stimulation of the sympathetic. But how is this stimulation produced ? Two hypotheses may be suggested : that of a neuritis of the sympathetic, or that of a reflex disorder involving the sympathetic paths.

It might be supposed, in the first place, that the wound has caused an irritative inflammatory process either in the sympathetic filaments accompanying the nerves of the cerebro-spinal system or in the peri-arterial plexuses which form the most important centrifugal sympathetic tracts. It is possible that this is so in some cases, but this mechanism must be at least exceptional. Indeed, as we have seen in the paralysis and contractures which we are studying there are no grounds for supposing a lesion of the nerve trunks. As regards the arteries, sphygmometric examinations show that they are intact, and it is difficult to imagine a lesion of the periarterial sheath under such circumstances. An even more important argument, perhaps, is that the vaso-motor and thermal disturbance may extend above the lesion and reach, for example, the arm above the elbow, and even the shoulder in a case of lesion of the hand. Sometimes the other arm as well shows some vaso-motor changes and disturbance of sweat secretion.[1]

[1] The occurrence in the corresponding sound limb of nervous changes which had at first been localised in the wounded limb was particularly evident in an officer whom we recently showed at the Neurological Society (164), and whose case we briefly described above (*v.* p. 562). In the course of the subsequent discussion Jean

Plate VII

A. Contracture of the flexors of the hand with hyper-extension of the last three fingers, following a bullet wound of the forearm at the junction of the middle and lower thirds, with fracture of the radius. The loss of motor power is not complete, the patient has always been able to carry out some slight movements of abduction and flexion of the fingers, but as soon as he makes no further effort to straighten them, the hand resumes its ordinary attitude. The condition has not shown any considerable change for a year (November 1916). Very marked vaso-motor symptoms and hypothermia. Succulent appearance of hand. Mechanical hyperexcitability of the small muscles of the hand with slowness of the muscular contraction.

B. Contracture of the flexors of the hand with hyper-extension of the first two phalanges and flexion of the last, after a shell wound of the back of the hand at the second metacarpo-phalangeal joint followed by prolonged suppuration. The contracture of the flexors of the hand progressively increased five or six months after the wound.

The loss of power is not complete: extension of the wrist, adduction and abduction of the fingers and flexion of the last phalanges are still possible, but the movements are carried out feebly. The mobility of the thumb is unaffected. Hypothermia and vaso-motor symptoms are very marked; in the forearm the oscillations are weaker than on the sound side. Slight mechanical hyper-excitability of the muscles of the hand increasing considerably after application of moderate cold, which does not modify the muscular excitability of the other arm.

C. Contracture of the flexors of the hand with hyper-extension of the fingers, following a wound of the shoulder with fracture of the head of the humerus (the arm was immobilised in a sling for a month). The flexion contracture of the hand was, associated with a contracture of the forearm, which did not appear until four months after the wound, and was preceded by paresis of the arm. Hypotonus of the flexors of the fingers is very marked, giving rise to a subluxation of the phalanges (*v.* Fig. 8, p. 554).

General atrophy of the fingers and mechanical hyperexcitability of the small muscles of the hand noted June 1916.

PLATE VII

All these phenomena are difficult to reconcile with the hypothesis which we have just discussed, and fit in better with a mechanism of a reflex character. In further support of this idea we may invoke the changeable character of these phenomena, which are liable to alter from one moment to another, hyperthermia succeeding hypothermia, and the amplitude of the oscillations measured by the sphygmometer being greater on the side of the injury, where it had been much weaker a few minutes previously.

The source of this reflex stimulus can only be looked for in the irritation of centripetal filaments coming from the seat of the trauma. It is difficult to know whether these centripetal fibres belong to the cerebro-spinal or sympathetic system, and the question is of little importance considering the close relations between these two systems. On the other hand, the centrifugal path of the reflex, as regards the vaso-motor and thermal phenomena, is certainly provided by the sympathetic.

It is interesting to compare our cases with the old experiments of François-Franck. Owing to the striking resemblance between clinical and laboratory observations a fairly full account of these researches may be given (198).

Camus made the following remarks: "At the last meeting of the Neurological Society I said that the phenomena described by the name of reflex paralyses and contractures did not in the great majority of cases obey the laws of reflexes (law of symmetry, irradiation and generalisation). . . . The cases shown to-day by Drs. Babinski and Froment illustrate my objection: the phenomena on the one hand were first unilateral and then bilateral (law of symmetry); on the other hand they are more pronounced on the stimulated than on the other side, in conformity with what is seen in experiments. One must be guarded, however, in comparing this clinical picture with experimental observations; in the latter, indeed, the phenomena appear at once as the result of variations in the intensity of the stimulation. In the clinical cases, on the contrary, the passage of the symptoms to the limbs of the opposite side required two or three months, which indicates, without doubt, a much more complex process than that which occurs, for instance, in the decapitated frog."

Action of cold on the vessels. François-Franck's experiments.—" In a first series of experiments the hand was plunged into a jar containing water at 18° C. As the jar was placed in a large vessel, a tap could be opened when required and made to pour at once a large quantity of very cold water on the jar, and consequently cause a considerable reduction in the temperature of its contents.

" Under these conditions constriction of the vessels of the hand was somewhat late in its appearance on account of the relative slowness of the fall of the temperature of the fluid in which the hand was placed, but there was an obvious diminution in size which became more pronounced as the chilling of the water increased. A fall of three or four degrees was enough to produce a very marked effect.

" I obtained similar but more decided results by chilling the water containing the hand in a different manner. . . .

" In order to investigate further the probable mechanism of the phenomenon, I carried out an experiment on the skin of the forearm instead of on the hand. I applied a large piece of ice to the skin on the antero-internal aspect of the elbow, while the lower part of the forearm and hand was plunged into the apparatus and a record was taken. . . .

" It was some little time before the vessels contracted; they remained contracted for a few seconds, and then gradually became dilated.

" Is not this the usual course of the phenomena of movement caused by a stimulus to the nerves? *A latent interval, a period of increase, and a period of decline or relaxation.*

" Let us now examine the probable course of this nervous action.

" The ice was applied to an area in which the collection of brachial nerves and vessels is very superficial. It might be supposed that the vascular nerve fibres accompanying the brachial artery and those contained in the median, internal cutaneous and ulnar nerves might be directly affected owing to their proximity to the point of application of the ice.

" In order to verify this hypothesis I resumed the experiment by applying the ice to the outer and posterior surface

of this region. The same effect was produced in spite of the thickness of the tissue separating the ice from the main body of vessels and nerves.

"I had, therefore, to think of the possibility of a reflex influence whose starting-point was in the *cutaneous nerve filaments* of the region on which the ice was applied, whose point of reflexion was in *the spinal nerve centres*, and whose destination was the *vascular nerves* of the limb under experiment.

"There does not appear to me to be any doubt about the reality of these reflexes from the afferent nerves to the vascular nerves, if one confines one's attention to the effect of the vascular constriction, as shown by the diminution in the size of the left hand, when the right is submitted for a moment to the action of cold. There is no disputing the fact, and it is only its interpretation which is open to discussion, but, as I will endeavour to prove, the vascular reflex accounts for the phenomena, and as this mechanism has been proved in the case of the crossed effect, it appears to me that it ought to be admitted in that of the direct effect. . . .

. . . . . .

"This explanation, which had already been suggested by Brown-Séquard and Tholozan, has been adopted by physiologists, who have repeated their experiments with success. But one must consider (1) whether the anatomical data allow this interpretation of our results; (2) whether the different stages of the phenomena agree with the well-known experimental results of reflex actions.

"1. The vascular nerves of the upper limb leave the spinal cord between the third and seventh dorsal roots, pass up the sympathetic chain, and then become distributed among the mixed nerves of the brachial plexus and the nerve filaments surrounding the large arterial trunks at the root of the limb.

"If, therefore, the vascular nerves of the upper limb arise from the cord at the same time as the ordinary motor nerves, it is easy to understand that a peripheral stimulus passing up to the region of the spinal centres from which they arise may be reflected along these vascular nerves as

well as along the roots of the nerves destined for the striated muscles.

"2. The different stages of the experiment entirely agree with the hypothesis of a reflex action."

Apart from their duration, the experimental facts related by François-Franck are almost entirely the same as our clinical observations. The duration of the vascular spasm in our cases is at first sight surprising. But it should be noted that the vascular spasm is not constant, and that favourable external influences rapidly cause a relaxation or even a more or less prolonged cessation of the spasm of the vessel wall.

We have now to examine the *consequences of this decline in the activity of the circulation on the working of the muscular system.* Our clinical study has shown that the vaso-motor and thermal disturbances are responsible, in great part at least, for the mechanical hyperexcitability of the muscles and the slowness of the muscular contraction. Do these results agree with what physiology teaches us ? Marey has shown that the muscular contraction diminishes in length and increases in amplitude as the result of heat, and on the contrary increases in length and diminishes in amplitude under the influence of cold and anæmia.

It appears at first sight as if there was a certain contradiction between the clinical cases which we are studying and the results of experiments, since with a hypothermia of 8° C. as indicated by the pyrometer in the temperature of the affected side as compared with that of the sound side, even a slight mechanical stimulus causes a contraction of muscle, not only slow and prolonged, but even of great amplitude.

As a matter of fact the muscle then behaves as in the first stages of intoxication by veratrine, one of the muscle poisons which has been best studied by physiologists who have found certain resemblances between its mode of action and that of autogenous poisons. "With small doses of veratrine, or at the beginning of the action of larger doses," writes Mlle. Joteyko

(212), " the first effect of the drug is to increase the muscular excitability. Carvallo and Weiss state that the contractions may be six times as high as normally."

Fatigue is followed by " a preliminary stage in which the contractions increase in extent and duration." They continue to increase in duration, while they diminish progressively in extent.

From this it follows that the amplitude of the muscular contractions in our clinical cases is not physiologically paradoxical. It is only the intensity and duration of the amplitude that are somewhat surprising, but this can be explained by the persistence of the vaso-motor and thermal disturbance.

Considering the very complex mechanism of heat regulation in organisms which are homoiothermic, or to use C. Richet's expression " strongly exothermic," the disturbance of muscular excitability should have a tendency not only to persist but even to increase under the accumulating influence of a series of causes when the atmospheric temperature is not favourable to those organisms.

" The perfection of movement," writes Richet (241), " is doubtless connected with a certain rise of temperature which is necessary to produce rapid and complete chemical action. Moreover, by a sort of marvellous cycle the intensity of the chemical reaction entails a more rapid production of heat. There is a sort of automatic adjustment, which regulates the rapidity and precision of movements (connected with a high temperature) by the very movement which raises the temperature."

The superiority of homoiothermic animals, which makes them independent of the surrounding temperature, finds an inevitable counterpart in the delicacy of their constitution. " The existence of very strongly exothermic animals," adds Richet, " is undoubtedly more fragile and more exposed to attacks of all kinds than that of cold-blooded creatures. The tissues are more eager for oxygen and materials of repair, and are therefore liable to die more readily. . . . Vigour and

Plate VIII

A. Flaccid paresis of the great toe with extension contracture of the other toes, following a bullet wound of the left ankle without fracture (September 1914). The contracture of the toes was associated with a contracture of the foot at a right angle. The contractures of the foot and toes do not disappear until an advanced stage of chloroform narcosis, and reappear at the same time as the tendon reflexes long before the return of consciousness (*v.* Appendix, Case XI, p. 665). We have had the opportunity of seeing two other wounded men with the same rare contracture.

B. Paresis of the dorsi-flexors of the left foot and extensors and flexors of the toes, following a wound of the gluteal region without a lesion of the sciatic nerve. Claudication of the same type as that shown in Pl. II, p. 504

When the patient tries to carry out a movement of dorsi-flexion of the foot, he can only raise the inner border of the foot which assumes a varus position. The movement of extension of the toes which is associated with dorsi-flexion of the sound foot does not occur on the affected side. The left knee jerk is brisker than the right; the left ankle jerk on the other hand appears weak, its movements being slow and of small amplitude. Exercise and artificial heating of the foot increase the amplitude to some extent and also improve its mobility.

Marked hypothermia and vaso-motor symptoms. Very pronounced mechanical hyperexcitability of the muscles of the foot and antero-external group of the leg.

C. Paresis of the dorsi-flexors of the right foot and extensors and flexors of the small toes, following shrapnel wound of the dorsum of the foot and leg complicated by fracture of the tibia (September 1915). The wound of the leg took five months to cicatrise, and the patient wore a walking apparatus for three months.

In April 1916 the loss of power is not complete, the movements of the great toe are preserved, the dorsi-flexion of the foot is chiefly carried out by the anterior tibial muscle, and is accompanied by a movement of adduction of the foot, which assumes a varus attitude; during this movement the outer toes remain flexed. Marked atrophy (five centimetres in the leg and seven centimetres in the thigh); hypothermia; mechanical hyperexcitability of the muscles of the foot, leg and quadriceps; crowding of the toes resembling the arrangement of the fingers so frequently seen in accoucheur's hand.

In June 1916 dorsi-flexion of the foot is carried out better, and the outer toes can be extended. The patient has been treated by diathermia and movements.

PLATE VIII

perfection in an organ are contradictory qualities, and we ought perhaps to regard those organs as the most marvellous which by reason of their progressive complexity have become susceptible to the least disturbing influences."

These physiological principles help to explain the clinical facts to which we have drawn attention, especially the persistence of reflex paralyses and contractures. They even serve perhaps to throw some light on hypotonus, as it is a fact that under the influence of cold and fatigue muscle fibres lose their elasticity and become lengthened (Boudet) (190).

We may also mention the observation of Schiff, who found in the frog after hibernation a state of latent contracture probably due to the stimulation of the muscles by the products of muscular katabolism. Possibly some forms of reflex contracture or hypertonus have a phenomenon of this kind as their immediate cause.

To sum up, the "reflex" pathogeny appears to us to explain all the peculiarities of the syndrome which we have described. Among the symptoms which constitute it, some, such as exaggeration of the tendon reflexes and vascular spasm are the direct result of a reflex action, while others, such as mechanical hyperexcitability of the muscles and slowness of their contraction, appear to be only an indirect consequence.

Lastly, it is possible that the motor disorders may depend, either as Charcot had supposed on a state of excitement or stupor of the spinal motor centres, or on disturbance due to sympathetic vaso-motor phenomena which are themselves of reflex origin.

The former mechanism doubtless explains the contractures with obvious or latent exaggeration of the tendon reflexes, such as some flexion contractures of the leg on the thigh or some contractures of the flexors of the forearm. The latter mechanism seems to account for those paretic or hypertonic states which are frequently seen to predominate in the extremities and to be accompanied by very marked vaso-motor disturb-

ance. Both mechanisms, however, often seem to be co-operating simultaneously, which accounts for the frequent association of symptoms found in both groups, *e. g.* in cases of contracture of the pelvi-trochanteric muscles with a paretic state of the foot.

But in spite of the value of the arguments in favour of a reflex pathogeny, some neurologists refuse to accept it, and in explanation of the facts to which they consent to give a certain individuality, they bring forward again the old interpretations which were so judiciously criticised by Vulpian and Charcot long ago. As in the past, some writers (Camus, Claude, Sollier, A. Thomas (140)) regard immobilisation as the cause of these symptoms, while others (Guillain and Barré (141)), attribute it to an ascending or radiating neuritis. It is a strange thing, it may be said in passing, that the problems, whose solution appears to be so simple, should still be under discussion, after having been advanced three-quarters of a century ago.

According to some, then, *immobilisation* should be incriminated; mere inactivity, they think, resulting from an hysterical paralysis or contracture can cause the various symptoms which they have described. This opinion seems to us to be erroneous. We do not dispute the fact that the immobilisation of a limb may cause a slight degree of local hypothermia, as thermo-asymmetry may be made to appear by making one arm move and leaving the other at rest. It is also justifiable to suppose that immobilisation is, to a certain extent, able to maintain and aggravate the vaso-motor and thermal symptoms which depend on reflex nervous disturbance, a fact which explains the therapeutic results obtained in some cases by active or passive exercises. What we deny is that the immobility by itself resulting from a purely hysterical contracture or paralysis is able to create definite vaso-motor and thermal disturbance, as well as all the other symptoms which we have described. Our opinion is not the outcome of an a priori theory, but is based on the

observation of a large number of hysterical contractures and paralyses, some of which are of very old standing.

In order, however, that the point should be settled scientifically once and for all, we invited all the neurologists at the Neurological Society on May 4, 1916, to make a collective investigation, definitely stating the conditions required to avoid confusion.

It was advisable, we declared, to make an enquiry similar to those which one of us recently instituted as to the disturbances of the tendon reflexes and pupillary reflexes, as well as to œdema, and especially blue œdema.

Only the pure cases were to be discussed, *i. e.*, those in which immobilisation was not associated with any other factor. No notice was to be taken of hysterical paralysis associated with a lesion (wound or arthritis) of the affected limb, for there would be no means of distinguishing what was due to immobilisation from what was due to reflex nervous disturbance. It would be illogical to use such cases for trying to prove that immobilisation was able to produce the syndrome in question. Every case was also to be rejected in which immobilisation was the result of an affection of the central nervous system, as such lesions were able to produce disorders of the vaso-motor and thermal mechanism and consequently all the nervous manifestations due to this cause. There would, therefore, be no justification for attributing such disorders to immobilisation. We may observe, however, that we examined from this point of view several persons suffering from organic paralysis who had been immobilised and bedridden for several years, but did not succeed in finding the slightest trace of vaso-motor and thermal troubles or of mechanical hyperexcitability of the nerves and muscles.

Further, no case could be considered in which the immobilisation was the result of a lesion to a nerve, even if the symptoms attributed to inactivity had been observed in an area with a different nerve supply. It is a well-known fact that in such cases nervous symptoms may be observed outside the area of the affected

nerve, *e.g.* the knee jerk may be exaggerated in sciatic neuritis, as one of us has shown (181).

Lastly, all cases of immobilisation in a plaster apparatus would have to be eliminated, as this is liable to cause compression and more or less persistent disturbances of nutrition and circulation.

It should be noted, however, that the femur may be recalcified in hip disease when the joint-lesion has healed in spite of immobilisation in plaster. A recent work by Ménard contains a series of skiagrams which have been taken in cases throughout their treatment by immobilisation, and which are very instructive in this respect. We will quote from it the following remarks on muscular atrophy in hip disease, and on the dystrophic influence of immobilisation to which in Ménard's opinion an excessive importance was frequently attached (228)—

"The muscles of all the segments of the lower limb undergo atrophy. This atrophy which was already obvious at the commencement of the affection increases as the tuberculous disease of the hip progresses. It has been said that the atrophy is increased by excessive prolongation of strict immobilisation. The fact is certain, but is of secondary importance. There is no doubt that the persistence of the joint lesion is the principal and essential if not the exclusive cause of muscular atrophy. The atrophy disappears more or less rapidly if the hip disease ends in recovery."

We will remind the reader that Charcot and Vulpian in combating the theory of immobilisation had urged that the so-called reflex atrophy may develop very shortly after traumatism.

The same may also be said of vaso-motor and reflex troubles : quite recently we saw an officer with a wound in the foot only a fortnight old without any lesion of the nerves or arteries who already showed very marked hypothermia of the foot and leg with hypæsthesia and considerable diminution of the cutaneous plantar reflex, though he had kept on his feet all the time.

Our views are supported by the observations of

Clovis Vincent (163), which recently formed the subject of a communication entitled "The prognosis of reflex nervous disorders. Persistence or increase of vaso-motor troubles and amyotrophy in spite of active and prolonged exercise of the affected limb." We are indebted to the writer for the following abstract of this work—

"The following is a short account of two cases which we had the opportunity of observing. The first case was that of a man who had been wounded in the foot without much injury to the bone. He came to consult me for the first time in July 1915, complaining of pain in the foot and walking on crutches. On examination the left calf was smaller than the right, the difference being four centimetres. The tendon reflexes were not affected, and there was no disturbance of the electrical reactions. There did not appear to be any relation between the disordered gait and the organic condition. Many of the symptoms appeared to be of a pithiatic nature. In fact, the treatment which I adopted soon enabled him to give up his crutches and walk by himself, with a slight limp, it is true. He was transferred to the auxiliary service.

"The pain gradually got worse and the limping increased. The patient was unable to carry out any duties, and was sent to the neurological centre at Montpellier, whence he was transferred to the neurological centre at Tours in September 1916. It should be noted that he had never been laid up, and that he had not ceased to walk daily with only one stick. When I saw him again the disturbance of his gait was very pronounced, and he declared that he was suffering considerably. The difference between the two calves was eight centimetres, and the thigh was atrophied, though it was not so in July 1915. There was mechanical hyperexcitability of the gastrocnemii. The right foot was much colder than the left.

"Thus, in July 1915 the pithiatic phenomena were as pronounced as the reflex ones, whereas in July 1916 the reflex phenomena were practically the only ones present, and necessitated his discharge from the army.

" In the second case in which the patient's daily exercise was under our own supervision, and was for a long time particularly applied to the affected limb, in spite of eight months' efforts, the muscular atrophy and vaso-motor troubles showed no obvious improvement.

" The case was that of a young man who received a superficial wound of the left knee in August 1914. In August 1915 I found the following symptoms : amyotrophy of the left calf, the circumference of which was 2·5 centimetres less than that of the right, the left ankle jerk weak and sluggish, premature fusion of the faradic contraction in the left gastrocnemius, and cyanosis and hypothermia of the affected foot, which was paretic, the voluntary movements being weak and limited, with slight flexion contracture of the leg on the thigh.

" For the eight months following this examination the patient was given an intensive re-education at the neurological centre at Tours. For two hours every day he was exercised in walking, running and hopping on the left lower limb. In September 1916, after twelve months' training, I found a certain improvement in the symptoms, the leg was now completely extended on the thigh, and the movements of the affected foot were almost normal, but the amyotrophy, and vaso-motor and electrical disturbances persisted without any change. The man recognised that his condition had improved, and had even undergone a complete transformation, but he could not walk more than four or five kilometres without much fatigue.

" I could quote many more examples of this kind. It must be admitted that whatever view be held as to the mechanism of these phenomena, whenever the so-called reflex symptoms have developed (I have in mind especially the vaso-motor troubles and muscular atrophy), exercise, however intense and prolonged it may be, cannot disperse or even improve them. Sometimes they actually become worse in spite of active exercise daily.

" I do not mean to say that men with reflex troubles should not undergo exercises and training. But one should know what results to expect. If the reflex troubles are slight and the hysterical phenomena associated with them

are severe, a successful result may be obtained, and in some cases the man may be able to return to duty. If, on the contrary, the reflex troubles are severe, no result will be obtained, however persistent one may be in training and re-educating these men, and they will have to be discharged from the service.

" Every week I have soldiers with severe reflex disorders sent to my service at Tours for re-education. I can do nothing for them, any more than any other doctor. Whenever I can do any good, the reason is that the associated hysterical phenomena are much more pronounced than the organic."

To sum up, from cases observed hitherto one is justified in concluding that traumatism may be followed by the appearance of vaso-motor and thermal disorders which may attain a high degree of intensity, and be accompanied by neuro-muscular and mechanical excitability as well as by hypotonus. It may be asserted that these troubles are liable to develop without there having been any immobilisation properly so called at any time. On the other hand, there is no conclusive case to justify the assertion that immobilisation by itself, such as may result from a pure and non-traumatic hysterical paralysis, can give rise to phenomena of this kind.

The second hypothesis which attributes the symptoms under consideration to an *ascending neuritis* has not been supported by any new argument, and the refutations of Charcot and Vulpian still hold good.

Pain along the course of the nerves, which was described by earlier writers, and on which Guillain bases his diagnosis does not by any means constitute a proof of neuritis : it may often, indeed, be very slight or entirely absent.

The hypothesis of an ascending neuritis—an affection imperfectly defined—is hardly suitable for nervous disorders which, in the first place, do not properly speaking take an ascending course, and, secondly, are not strictly confined at any period of their development

to a peripheral nerve area. To justify such a diagnosis, it would be logically necessary for the disorders to be primarily localised in one of the terminal branches of a nerve and to extend progressively to the collateral branches situated at higher levels.

Such is also the opinion recently expressed by Gougerot and Charpentier (136): "Ascending neuralgia and especially ascending neuritis corresponding to the text-book description, appear to be rare (we have not met with a single case of proved ascending neuritis), whereas reflex paralysis and trophic disorders accompanied by ascending neuralgia are frequent. Although at first; owing to the want of a better term and our ignorance of recent observations, we were inclined, like other medical men, to label several of these reflex disorders ascending neuritis, the two classes of phenomena are quite distinct, and we are inclined to think that a certain number of cases formerly diagnosed as 'ascending neuralgia and neuritis' were really instances of the recently described cases of reflex disorders."

Sicard writes in the same strain (149): "It is interesting to note that in a considerable number of persons whom we have examined suffering from various wounds of the peripheral nerves, *we were unable to find a single case of ascending neuritis.*

"This observation tallies with the conclusions which we expressed some time ago in a report on ascending neuritis, in which we protested against the prevalent conception of this syndrome."

This pathogeny can apply at most to only a very small number of cases. But to accept it in any given case, in the absence of characteristic clinical signs of neuritis, such as reaction of degeneration or loss of the tendon reflexes, it would be necessary to have proofs based on anatomical examinations. Obviously it would not be enough to find nerve fibres showing pathological changes in the affected area, for their existence is a matter of course, but it would be necessary to find undoubted lesions in the nerve trunks to explain the symptoms observed.

It is also to be noted that the exaggeration of the tendon reflexes, which is fairly common in reflex nervous disorders, is opposed to the hypothesis of a neuritis, an affection in which the tendon reflexes are generally diminished or abolished. If a neuritis can cause exaggeration of the tendon reflexes in the area of the damaged nerves, which is by no means proved, it occurs in very exceptional cases only.

One last remark before concluding this study in pathogeny. Some of the clinical forms described in the chapter on "Symptomatology" by the name of congealed hand, accoucheur's hand, or acro-contracture present, as we have seen, a striking analogy with the types of contracture observed in attacks of *tetany*.

We may also add that, as in reflex contractures and paralysis, cases of tetany have been seen with increase in the mechanical excitability of motor nerves (Chvostek's phenomenon), increase in the electrical excitability of nerves (Erb's phenomenon), changes in the tendon reflexes, which are sometimes weakened and sometimes abolished, vaso-motor disorders and muscular atrophy.

These resemblances between the so-called reflex contractures and the contractures of tetany, which are unanimously regarded as due to a stimulation of the nerve centres by a poison impregnating them, constitute a fresh argument against the conception that the so-called reflex nervous disorders are due to mere immobilisation. They tend also to show that these contractures are not the direct consequence of peripheral lesions produced by traumatism, but that they imply a disturbance of the nerve centres, and this is, after all, the principal idea implied by the "reflex" pathogeny.

Lastly, we may note that there is no need to oppose the "reflex" pathogeny to the "sympathetic" pathogeny, as some have done. On the contrary, they harmonise with one another very well, the vaso-constriction being the result of a reflex action exercised through the sympathetic system.

It is not surprising that symptoms of a reflex character should result from a peripheral lesion which causes disturbance in the spinal centres and in the sympathetic system simultaneously. To understand this it is sufficient to quote the following passage from François-Franck's lectures—

"The vascular nerves of the upper limb leave the cord between the third and seventh dorsal roots, pass up by the sympathetic chain, and then become distributed among the mixed nerves of the brachial plexus and the nerve filaments surrounding the large arterial trunks at the root of the limb. If, then, the vascular nerves of the upper limb come from the spinal cord at the same time as the ordinary motor nerves, it is easy to understand how a peripheral stimulus passing up to the region of the spinal centres from which they emanate may be reflected along these vascular nerves as well as along the roots of those nerves destined for the striated muscles."

PART III

PITHIATISM AND REFLEX NERVOUS DISORDERS

CHAPTER IX

DIAGNOSIS

Hysteria has been and still is so frequently confounded with reflex nervous disorders, that we have thought it advisable to collect in one part of the book everything relating to the diagnosis of these two groups of nervous manifestations; the difference between them will thus be more clearly illustrated.

The first section will be devoted to the diagnosis of hysteria and organic affections of the nervous system, but we shall deal with only a part of the problems relating to this question. Hysteria undoubtedly may be mistaken for any of the affections of the nervous system, since it is capable of imitating them all. But in consideration of the restricted scope of this work we will devote ourselves to the study of the clinical problems raised by the different types of paralysis and contracture so frequently found in the neurology of war. We have already had occasion to describe the diagnostic features of a convulsive attack (*v.* p. 456), mutism (*v.* p. 485), deafness (p. 480), and hysterical pseudo-sciatica (p. 462), to mention only the most frequent pithiatic phenomena. In the following section we shall set forth the differential diagnosis between reflex nervous disorders and organic affections of the nervous system. We shall then do the same with

hysteria and reflex nervous disorders. Lastly, we shall show how hysteria can be distinguished from simulation, but we shall first deal with those morbid phenomena which are so important in neurology and especially in the neurology of war.

The Diagnosis of Hysterical Paralysis and Organic Paralysis

Some hysterical manifestations are quite characteristic, e. g., *hysteria major*, *rhythmic chorea*, and *mutism*. No nervous disturbance due to an organic affection resembles them; they can therefore be recognised at first sight. It is also the same with *certain types of systematised paralysis*. For a paralysis to be placed in this group it is not necessary for some of the motor functions of the limb to be completely abolished, and the others to be completely preserved. All that is required is that there should be a considerable difference in the activity in the same group of muscles according to the movements which it has to perform.

Such a variety of paralysis cannot invariably be regarded as hysterical in nature. It seems that cerebral lesions may cause changes in the movements of the eyeball analogous to the paralysis which we are considering. As regards the limbs, the so-called functional spasms, which are very resistant to treatment and have absolutely nothing to do with hysteria, are also motor disorders which only affect certain acts. It is none the less true that no functional spasm or any organic affection reproduces any of the following types of systematic paralysis, which are characteristic of hysteria. Take, for instance, the case of a wounded man whose inability to stand upright or to walk is in contrast with the more or less complete freedom of the movements of the lower limbs when lying down. There is no doubt that this is an example of *hysterical astasia-abasia*. The diagnosis of pithiatism can also be made at once when an almost complete inability to carry out voluntarily in bed the elementary movements of

flexion and extension of the toes, foot, and leg co-exists with the ability to stand upright and walk a few steps. A facial paralysis, which is shown only in the unilateral movements of one of the buccal commissures (the acts of whistling or blowing being normal), also bears the stamp of an hysterical manifestation.

The *variability* of a contracture or paralysis may also be of significance in certain circumstances. Hysterical paralyses and contractures are sometimes modified in their intensity and form not only from one day to another, but even from one moment to another. These variations can easily be shown by certain tests. If an attempt be made to overcome a purely hysterical contracture, it will be found that the resistance is intermittent; in the course of the attempt the patient gives way, then recovers, and finally either diminishes or increases his contracture. Here is another patient with an hysterical flexion contracture of the last three fingers resembling a clawhand, due to a lesion of the ulnar nerve. If he is blindfolded to prevent him determining the exact position of his fingers, and he is then ordered to perform repeated, symmetrical and exactly synchronous movements with his hands, it will be found that the amplitude and form of the movements carried out by the contractured fingers vary considerably from one moment to another, an occurrence which does not take place in a patient with organic contracture.

Transitory disappearance is still more characteristic. Take, for instance, two cases, one with organic hemiplegia, and the other with hysterical hemiplegia, both of whom appear to be incapable of carrying out any movement whatever in the different segments of the paralysed upper limb. Try the following experiment: seize the paralysed arm, raise it and then leave it to itself, repeat this operation several times in succession, and at the same time attempt to distract the patient's attention from what you are doing by your questions. You will find that in the first patient the paralysed arm will drop down at once like a lifeless object as soon

as you have let it go, and this phenomenon will recur whenever you repeat the attempt. In the hysterical patient you will generally find the same thing in most of your experiments, but it may occasionally happen that the raised limb keeps the position you have given it for some time after you have let it go. The paralysis will have temporarily disappeared. This characteristic, which is always absent in organic hemiplegia, is of the utmost importance from the diagnostic point of view.

The following test affords a further illustration. Support the paralysed limb with the hand and raise it to a certain height; the inert limb, in obedience to the laws of gravity, will give the hand on which it rests a feeling of weight, and will produce a static effort which will always be the same in the case of the patient with organic hemiplegia, while on the contrary in hysteria it will be liable to variations, and may even completely disappear for some time (25).

It is also the same with pure hysterical contractures, which we have always regarded as essentially intermittent. If a sudden examination is made of a limb affected with this sort of contracture, and which had formerly resisted every effort to overcome it, the vicious attitude is sometimes reduced without using any force. This test requires a certain rapidity and abruptness, so that the patient has no time to collect himself: a sudden attack, so to speak, is made upon him. For this reason our friends are in the habit of giving the name of "the sign of the panther" to the correction of the vicious attitude obtained by this method. It may even be possible to see the contractured tendons and muscles stand out under the skin, when we come near the patient, as if he were on his guard, and then to notice that they relax as soon as we go away or another limb is chosen for examination.

All these intrinsic characters give hysterical contractures and paralyses an individuality which enable them to be recognised with almost complete certainty, but

it is not always possible to find them. What other means are available for establishing a diagnosis of hysteria in their absence ?

In the course of this work we have repeatedly shown that owing to the frequent association of hysteria and organic disease it was impossible to attach much importance to the presence or absence of stigmata. We will not return to this question, which has already been sufficiently discussed (*v.* p. 454).

In our opinion it is impossible to be guided by the existence of an *interval* between the occurrence of the trauma and the appearance of the nervous symptoms. There are, in fact, organic nervous symptoms which do not develop until some time after the trauma which caused them. This is frequently the case in hæmatomyelia, due to a shell explosion—a wounded man being able to walk to the aid post four or five hundred yards away, but as soon as he gets there he will be struck down by paraplegia and be unable to move from the stretcher. The interval is not always so short; in a case of hæmatomyelia of the same origin, motor symptoms consisting in quadriplegia did not appear till three days after the trauma, and were at first preceded by a simple asthenic condition, which allowed the patient to join up with his company (Léri, Froment and Mahar (222)).

Thus the hysterical nature of a phenomenon cannot be established by the prodromal circumstances, the existence of an interval, or even by neuropathic antecedents. At most they help to form a presumption which is often misleading. To avoid mistakes it seems best not to attach much importance to them.

In dealing with a nervous disorder which does not possess those characteristic features which justify its being attributed at once to hysteria, the diagnosis can be made only by exclusion. It can be established only by negative characters, or *the absence of the objective signs which characterise organic or physiopathic affections of the nervous system*, and whose value has been definitely settled.

It should be stated, however, that though the presence of such signs may enable one to establish with certainty the organic nature of a paralysis or contracture, their absence does not allow us to assert categorically that it is a case of hysteria, though such a diagnosis is very probable. There are, indeed, good reasons for supposing that in addition to the objective signs which we know there are others unknown to us at present, the discovery of which will one day enable us to separate from hysteria those conditions at present attributed to it.

In discussing the problems in diagnosis which occur most frequently in medical practice, we will describe the objective signs which have been in part discovered or recommended by one of us (61), and enable a distinction to be made between organic and hysterical paralysis.

Organic Hemiplegia and Hysterical Hemiplegia

We have drawn up a table of the motor symptoms, distinguishing organic hemiplegia from hysterical hemiplegia (25).

Organic hemiplegia.	*Hysterical hemiplegia.*
1. The paralysis is limited to one side of the body.	1. The paralysis is not always limited to one side of the body. This particularly applies to paralysis of the face, in which the symptoms are generally bilateral.
2. The paralysis is *not systematic*, e. g., if unilateral movements of the face are very much weakened, this weakness appears quite as distinctly on the hemiplegic side during the execution of	2. *The paralysis is sometimes systematic.* It is almost always so in the face, *e. g.*, unilateral movements of the face may be completely abolished, while the muscles on the hemiplegic side act nor-

synergic bilateral movements.

3. The paralysis affects the conscious and voluntary movements as much as the unconscious or subconscious voluntary movements, giving rise to the *platysma sign* (more energetic contraction of the platysma on the sound side in the act of opening the mouth or bending the head in opposition to the resistance which the observer makes to this movement), *combined flexion of the thigh and trunk*, and in walking the *absence of active swinging of the arm*, as contrasted with the exaggeration of passive swinging (the limb swaying to and fro like a lifeless object when the patient suddenly turns round).

4. The tongue is usually slightly deviated to the side of the paralysis.

5. There is, chiefly at the onset, muscular hypotonus, which may be shown in the face by obliteration of the naso-labial fold and the lower-

mally during the execution of synergic bilateral movements.

3. The unconscious or subconscious voluntary movements are not affected; with the result that there is an absence of the platysma sign, and of combined flexion of the thigh and trunk; active swinging of the arm may be absent, but there is no exaggeration of the passive movement.

4. The tongue is sometimes slightly deviated to the side of the paralysis, but the deviation may be very pronounced or be directed to the opposite side to the paralysis.

5. There is no muscular hypotonus. When there is facial asymmetry, it will be found to be due not to muscular hypotonus, but to spasm; the sign of ex-

Signs of Organic Hemiplegia

Fig. 16.—Normal plantar cutaneous reflex.

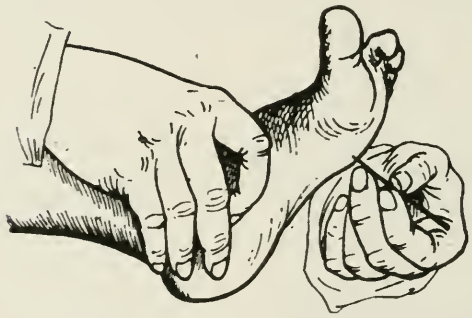


Fig. 17.—Extensor response (Babinski) (165, 167).

Fig. 18.—Exaggerated flexion of the forearm in right hemiplegia (Babinski) (166).

SIGNS OF ORGANIC HEMIPLEGIA (*continued*).

FIG. 19.—Combined flexion of the thigh and trunk in left hemiplegia (Babinski) (168).

FIG. 20.—Platysma sign in left hemiplegia.

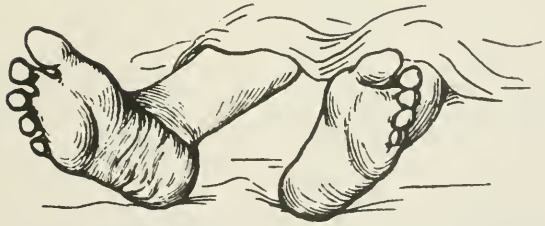


FIG. 21.—Abduction of the toes associated with combined flexion of the thigh and trunk in right hemiplegia (Babinski) (171).

ing of the eyebrow, and in the upper limb by *exaggerated passive flexion of the forearm*, and by the *sign of pronation* (the hand when left to itself assumes a position of pronation).

6. The tendon and bone reflexes are frequently affected at the onset, when they may be lost, diminished or exaggerated. Later, they are almost always exaggerated, and in many cases there is ankle clonus.

7. The cutaneous reflexes are generally affected.

The abdominal reflex and cremasteric reflex are usually diminished or lost, especially at first.

The character of the reflex movement of the toes following stimulation of the sole usually undergoes inversion; the toes, and especially the great toe, instead of being flexed, become extended on the metatarsus (*toe phenomenon*).

Extension of the great toe is often associated with abduction of the other toes (*fan sign*).

Exaggeration of the reflexes of defence may sometimes be noted.

aggerated flexion of the forearm and the sign of pronation are absent.

6. The tendon and bone reflexes show no change: there is no ankle clonus.

7. The cutaneous reflexes do not appear to be affected.

The abdominal and cremasteric reflexes are normal.

The reflex movement following stimulation of the sole does not undergo inversion. The toe phenomenon and fan sign are absent.

The reflexes of defence are not exaggerated.

8. The form of contracture has particular characteristics and cannot be reproduced by a voluntary contraction of the muscles.

There is a "clawing" of the hand, which gives the sensation of an elastic resistance automatically increased during passive movements of extension of the fingers.

9. The course is regular, contracture succeeding flaccidity. The disappearance of the motor disturbance, when it does take place, is progressive.

The paralysis shows no tendency to become better and worse alternately (*permanence of motor troubles*).

8. The form of contracture may be reproduced by a voluntary contraction of the muscles.

9. The course is capricious; the paralysis may remain flaccid indefinitely, or it may be spastic from the first; spastic phenomena are sometimes associated with paralysis, especially in the face.

The symptoms are frequently liable to subside and to get worse alternately, to become rapidly modified in their intensity as well as in their form, and to present transitory remissions which may last only a few moments (*variability of motor troubles*).

In addition to the above, other signs of organic hemiplegia have been described, of which we will mention the following—

10. *Raimiste's sign* (239), which is observed in the period of flaccidity. It is obtained as follows: Place the paralysed forearm and hand in a vertical position, with the elbow resting on a table. It will be found

that if the hand be left to itself, it will become rapidly flexed and at the same time pronated.

11. The *interossei phenomenon* described by Souques (242): movement of extension and abduction of the fingers whenever the patient raises the affected arm.

12. *Klippel and Weil's sign* (214): involuntary flexion of the thumb accompanying passive straightening of the flexed fingers (in the period of contracture).

13. The *tibialis anticus phenomenon* (Strümpell): an associated movement of dorsal flexion and adduction of the foot caused by voluntary flexion of the affected limb.

14. *Associated adduction and abduction* of the paralysed lower limb (Raimiste) observed in the patient lying on his back when he makes an energetic effort to adduct or abduct the sound limb against resistance.

15. *Various associated movements* which according to P. Marie and Foix (227) may be divided into the three following classes: *general synkinesis* (general contraction of all the muscles of the hemiplegic side on the occurrence of any effort), *imitation synkinesis* (involuntary movements of the hemiplegic side tending to reproduce the movements carried out voluntarily by the sound side), and *co-ordination synkinesis* (voluntary contraction of certain muscular groups in the paralysed limb giving rise to involuntary contraction of the functionally synergic muscles).

16. *Neri's sign* (237): flexion of the knee, accompanying flexion of the trunk on the paralysed side.

17. The *dorsal reflex of Mendel-Bechterew* (185), or dorso-cuboid reflex: flexion of the toes of the paralysed subject produced by percussion of the latero-dorsal surface of the cuboid, an opposite movement to that which occurs normally. This interesting phenomenon is associated with exaggeration of the tendon reflexes.

18. *Reflex hyperkinesis* (Claude) (194). Painful stimulation by pricking, pinching, or pressure of the muscles sometimes causes reflex movements in the paralysed upper limb.

19. The *reflex of adduction of the foot* (Raichline, P. Marie and H. Meige) obtained by stimulation of the skin on the inner border of the foot.

It should be noted that none of these signs is constant; the majority even may be absent in ill-developed organic hemiplegia, especially, it appears, when there is a traumatic cortical lesion.

Organic Paraplegia and Hysterical Paraplegia

It is mainly by the *absence of the objective signs* peculiar to organic paraplegia that hysterical paraplegia is recognised. These signs vary according to the seat of the lesion. They are as follows—

1. *Changes in the tendon reflexes.*—If the lesion is in the dorsal region, there is generally an exaggeration of the tendon reflexes of the lower limbs, accompanied sometimes by ankle clonus and patellar clonus. If the lesion is in the cervical region, in addition to exaggeration of the tendon reflexes in the lower limbs, there may be modifications in the reflexes in the upper limbs. According to the position and extent of the anatomical changes the reflexes may be all exaggerated, diminished or lost, or some may be lost and the others preserved or exaggerated (inversion of the radius reflex, paradoxical reflex of the elbow). Lastly, if the lesion is in the lower part of the cord or in the cauda equina, the tendon reflexes of the lower limbs are generally weakened or abolished.

2. The *toe phenomenon*, a characteristic sign of a disturbance of the pyramidal tract.

3. *Exaggeration of the reflexes of defence* which has the same significance as the preceding symptom. It can be best exhibited as follows: The skin of the dorsal aspect of the foot or lower part of the leg is vigorously pinched. When stimulation of this kind produces *dorsal* flexion of the foot, one is justified in asserting that the reflexes of defence are exaggerated (180). This mode of reaction is never found when the pyramidal tract is intact. Normally, this movement can only be

caused by stimulation of the sole, and even then it is less extensive and less sustained than in pathological conditions.

4. *Amyotrophy with reaction of degeneration* when the grey substance of the anterior cornua or the anterior roots have been affected.

5. *Sphincter troubles.*

6. *Trophic cutaneous* changes, including bed-sores. All these signs are invariably absent in hysterical paraplegia.

Peripheral Neuritis and Hysterical Paralysis

Generally speaking, the diagnosis is easy. There are several obvious signs peculiar to neuritis, which are never found in hysterical paralysis, viz.—

1. *Diminution or abolition of the bone and tendon reflexes*, e. g., of the ankle jerk in lesions of the sciatic nerve, of the knee jerk in lesions of the anterior crural, of the reflex of flexion of the forearm in lesions of the musculo-cutaneous, and of the triceps in injuries to the upper part of the musculo-spiral. In the last case flexion of the forearm instead of extension is sometimes observed on percussion of the triceps tendon (paradoxical reflex of the elbow).

2. *Muscular atrophy*, which should be taken into consideration only when it is sufficiently pronounced, as slight amyotrophies may exceptionally be found in hysterical paralysis.

3. *Complete or partial reaction of degeneration.*—But it must not be forgotten that even in cases of nerve section it is barely established before the eighth or tenth day.

4. *Hypotonus.*

5. Exclusive localisation of motor troubles, degenerative amyotrophy and sensory troubles in an area exactly corresponding to the anatomical distribution of one or more nerves (*peripheral topography of motor, trophic and sensory troubles*).

The attitude of the limb affected by peripheral

paralysis and the changes which this causes in its ordinary movements are often sufficiently characteristic. The present circumstances have no doubt made certain deformities, *e. g.*, the wrist-drop of musculo-spiral paralysis and the clawhand of ulnar paralysis, an everyday affair, and given rise to hysterical paralyses which resemble them, but the imitation is never a close one.

It is possible, for instance, to simulate inability to extend the fingers on the hand, and the hand on the forearm, but a simulator is unable to dissociate purposely the contraction of the muscles of the anterior region of the arm (biceps and brachialis anticus) from that of the supinator longus, as is done involuntarily by a patient with musculo-spiral paralysis during an energetic movement of flexion at the elbow. The associated flexion of the wrist accompanying energetic flexion of the fingers is also an interesting feature, as one of us has shown; it indirectly reveals the relative weakness of the extensors and the predominance of the flexors. The importance of this symptom lies in the fact that it may be observed in patients who have only a slight lesion which does not prevent extension of the hand on the forearm.

The mere study of certain morphological peculiarities in the affected limb in repose and during various movements will enable one to recognise infallibly peripheral neuritis. We think it advisable to dwell on this question, which has been brought into prominence again by the neurology of war, and to show some definite examples of what may be regarded as characteristic changes in form. We shall then consider different movements of the hand and fingers in succession.

When the thumb is adducted and applied close to the outer border of the index it normally forms two obtuse angles of about 160°: the upper angle opens outwards, its sides corresponding to the outer border of the forearm and first metacarpal, while the lower angle opens inwards and faces the palm of the hand,

being formed by the first metacarpal, the first phalanx of the thumb, and the extended second phalanx.

This outline of the adducted thumb is altered in ulnar and median paralysis, but its change in appearance differs entirely according to which of the two nerves is involved by the lesion.

Fig. 22.—Paralysis of the right ulnar. When the patient adducts the thumb, the radio-metacarpal and metacarpo-phalangeal angles disappear on the side of the ulnar paralysis and the second phalanx of the thumb becomes flexed.

Fig. 23.—Paralysis of the right median, with adduction of the thumb. The radio-metacarpal and metacarpo-phalangeal angles are more pronounced and come nearer to a right angle on the side of the paralysis than on the sound side.

In paralysis of the ulnar, when the thumb is adducted, the metacarpal becomes flexed and the first phalanx extended, causing a disappearance of the two angles (the lower angle even tends to be sometimes hyperextended); on the other hand, the flexion of the second phalanx which always accompanies this kind of adduction leads to the formation of another angle (Figs. 22 and 24). This peculiar appearance is explained in cases of paralysis of the adductor pollicis by a supplementary adduction pro-

duced by the extensor longus, and which appears to involve the synergic contraction of the flexor longus pollicis.

In paralysis of the median the outline of the adducted thumb is quite different (*v.* Fig. 23). The second phalanx remains extended as in the normal condition, and instead of disappearing the two angles become more pronounced and come nearer to a right angle (predominant action of the adductors and extensors after paralysis of the adductor brevis).[1]

Abduction of the thumb is not affected in ulnar paralysis, but it is abnormal in paralysis of the median: the second phalanx of the thumb cannot be extended, and the metacarpo-phalangeal angle, which normally disappears in this movement, is almost as marked as in adduction. In musculo-spiral paralysis this movement is impossible, and is replaced by a movement of opposition either to the index or to the ring finger when the abductor brevis is paretic (as is sometimes seen in pure musculo-spiral paralysis).

Fig. 24.—Paralysis of the left ulnar: the patient is trying to bring the fingers together, but the left *little finger* remains *abducted*, and the second phalanx of the thumb becomes flexed as in Fig. 22.

[1] This characteristic deformity of the thumb in paralysis of the median nerve has been very fully described by Létievant (226), in whose *Traité des Sections Nerveuses* we recently found the following description: "Deformity of the thumb characterised by marked projection outwards of the head of the first metacarpal; a depression above corresponding to the atrophied abductor brevis and opponens pollicis, and an internal rotation of the whole thumb, caused by the contraction of the adductor so that its palmar surface looks directly forwards" (p. 442 *v.* also p. 443.

When a patient with paralysis of the median endeavours to make a movement of *opposition*, he can only carry out a pseudo-opposition : the thumb is not separated from the palm or is separated insufficiently, and does not face the hand ; it still keeps the same outline, but in addition to the upper and lower angles already mentioned there is a new angle caused by flexion of the last phalanx when the flexor longus is not paralysed.

As regards the other fingers, one may consider that *habitual abduction of the ring finger* with inability to adduct it, at least when the patient does not flex the

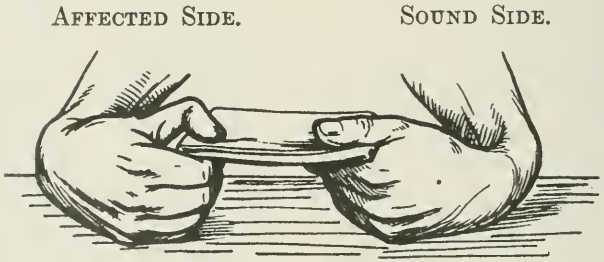


Fig. 25.—Thumb sign in paralysis of the right ulnar (Froment) (203), (204).

first phalanges, is one of the most constant and characteristic signs of ulnar paralysis (*v.* Fig. 24).

Lastly, it is often advisable in estimating the degree of mobility of a limb to make it execute *ordinary automatic* actions, such as *grasping movements.* In paralysis of the ulnar (with loss of power of the adductor) the vigorous grasp of a thin object, such as a newspaper or a piece of cardboard, between the thumb and index, bent beneath it, does not take place normally (first phalanx flexed, second phalanx extended or barely flexed, thumb approximated to the base of the index and directed towards its inner border). The thumb is doubled up and the grasp is made with the tip of the finger; the first phalanx is extended, the second flexed, and the thumb is often brought to the extremity of the index and directed towards its

outer border (*v.* Fig. 25). These peculiarities are explained by the adoption of another mode of prehension and by the predominant action of the abductor brevis. This is known as the *thumb sign* (Froment) (203).

In paralysis of the median, on the contrary, the deformity is best shown by grasping a rather thick object, like a cylinder or a glass, when the thumb strikes against the object and does not grasp it.

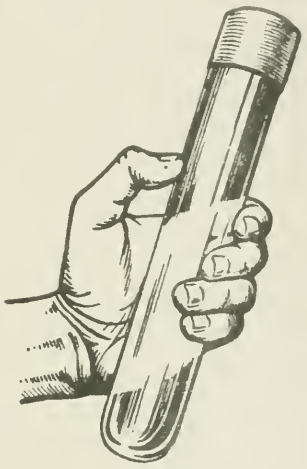


FIG. 26.—ABNORMAL GRASP IN A CASE OF LEFT MEDIAN PARALYSIS. Owing to the inability to oppose, the thumb is pushed against the cylinder and does not grasp it.

We may add that *unipolar faradisation* in paralysis of the median and ulnar may cause a *paradoxical extension* of the hand when the negative electrode is applied to the antero-inferior part of the forearm, whereas bipolar faradisation causes flexion. The latter indicates the predominant action of the extensors, and enables one to detect even a slight weakness of the flexors of the hand (183).

Unipolar faradisation of the postero-superior surface of the forearm causes, on the other hand, a *paradoxical flexion* of the hand in musculo-spiral paralysis even when the faradic excitability of the musculo-spiral nerve does not appear appreciably affected (184). The reason is that as the electrical stimulus is applied to the median and ulnar at the same time as the musculo-spiral, the action of the flexors is stronger than that of the extensors. This sign may serve to detect an incomplete musculo-spiral palsy, such as may occur from the use of crutches.

We may add that Raimiste's test for the diagnosis of organic hemiplegia may also be used for the diagnosis

of musculo-spiral paralysis (H. Meige, Froment and Hoven).

These diagnostic points, in addition to those which we have previously described, will always enable hysterical paralysis to be distinguished from paralysis due to a lesion of a nerve trunk.

Diagnosis of Reflex Paralysis from Organic Paralysis

Reflex paralyses, with or without contracture, are localised in the injured limb, and consequently never assume the form of hemiplegia or paraplegia, unless there are multiple traumata. There is thus a whole series of cases in which it is possible to eliminate at once the hypothesis of reflex paralysis. In this respect the diagnosis of reflex paralysis from organic paralysis is easier than that of hysterical paralysis from organic paralysis, or at least its field is more limited.

But, on the other hand, reflex paralyses and contractures may show exaggeration of the tendon reflexes, considerable amyotrophy, hypotonus, vasomotor and thermal disorders, changes in the electrical and mechanical excitability of the muscles, phenomena which belong to the symptomatology of the organic affections of the nervous system and are absent in hysterical paralysis. From this point of view the diagnosis is more difficult, and in some cases errors are more likely to occur.

After these preliminary remarks we will discuss the various problems which may arise.

Monoplegia associated with a Lesion of the Pyramidal Tract. Reflex Contracture and Paralysis

Apart from the etiological factors, which are entirely distinct in these two conditions and thus form a most important guide, the following form the data for diagnosis—

Organic monoplegia of central origin.	*Reflex contracture and paralysis.*
1. The paralysis often occupies the whole limb; it may be complete, either in the upper or lower limb.	1. The paralysis hardly ever occupies the whole limb. In the upper limb as a rule only the movements of the fingers and hand are affected, and as a rule the paralysis is not complete. In the lower limb the root of the limb is often affected, but here, too, the paralysis is only partial.
2. Flaccid paralysis which lasts only a few weeks is usually followed by contracture.	2. The paralysis may remain flaccid for a long time. It frequently coexists with contracture, the hypertonus and hypotonus occupying different muscular groups.
3. In paralysis with contracture flexion with clawhand is the type seen in the upper limb, and in the lower limb the extensor type; the patient circumducts his leg in walking (*helicopodal gait*).	3. The deformity of the upper limb in the hypertonic forms often presents a peculiar appearance, e.g., *accoucheur's hand*, *holy-water-vessel hand*, and *crowded fingers*. In regard to the lower limb, the attitude and gait vary, but the patient never circumducts his leg in walking.
4. The tendon reflexes are exaggerated some weeks after the onset of the paralysis. Exaggeration of the reflexes necessarily accompanies the contracture.	4. The condition of the tendon reflexes varies. Exaggeration is often absent, even in hypertonic forms.

5. The plantar cutaneous reflex is generally affected in crural monoplegia: the extensor response is found.

5. In paralysis of the lower limb the cutaneous reflex may be lost, but it reappears after the foot has been warmed; the extensor response is never found.

Peripheral Neuritis. Reflex Contracture and Paralysis

The etiological factors are the same in both conditions, if traumatic neuritis, which is the commonest in the neurology of war, is only considered. The following characteristics enable a distinction to be made between the two affections—

Peripheral neuritis.

1. It is the rule for the motor and sensory disturbances and degenerative amyotrophy to be localised in an area exactly corresponding to the anatomical distribution of one or more nerves (*neuritic topography*).

This is the cause of the deformities and attitudes described above.

2. *Amyotrophy is very pronounced.* The muscular prominences disappear, and certain regions of the limb become emaciated.

3. *Reaction of degeneration*, and especially great weakness or abolition of

Reflex contracture and paralysis.

1. The localisation of the motor and sensory disturbances and degenerative amyotrophy does not correspond to any anatomical area (*more or less segmentary topography*).

The deformities and attitudes are typical.

2. Amyotrophy variable, usually fairly marked, but never reaching the degree of intensity seen in neuritis.

3. *No reaction of degeneration.* The faradic muscular excitability

the faradic muscular excitability.	never shows marked weakening, but is often normal, and sometimes even exaggerated.
4. The tendon reflexes corresponding to the muscular distribution of the affected nerve are diminished or lost.	4. When the tendon reflexes are affected, they are generally exaggerated; they are never abolished.

Volkmann's Ischæmic Paralysis and Reflex Paralysis

Volkmann's ischæmic paralysis or contracture (189), an affection also called retractile myositis, which chiefly occurs in the child of five to ten years of age, but which may also be observed in the adult, generally follows the application of too tight a splint in fractures of the forearm. In typical cases the long flexors of the fingers and the pronators are affected. It is preceded by intense pain and œdema. It gives rise to a more or less marked limitation of the movements of the hand and fingers, fibrous retractions which form rapidly, sensory disorders of a segmentary distribution (glove anæsthesia), hypothermia, and occasionally a modification in the colour of the forearm (red coloration).

The symptomatology of this affection thus obviously resembles that of reflex paralyses and contractures in some respects.

The principal features by which the two conditions can be distinguished, apart from their etiology, are the board-like hardness of the muscles and the remarkable diminution, or even absence, of faradic and voltaic muscular excitability, which are present in Volkmann's ischæmic paralysis, and are absent in reflex paralysis and contractures.

Arterial Obliteration and Reflex Vascular Spasm

When arterial obliteration is accompanied by attacks of characteristic intermittent claudication and gangrene, or in those cases in which it is situated in the upper limb when it is revealed by a disappearance of the radial pulse—phenomena which are always absent in reflex spasm—the diagnosis is perfectly obvious. If, in addition to the vascular spasm, the motor disturbances previously described appear in a characteristic and fully developed form, no mistake can possibly be made : the case is one of reflex nervous disorder, arterial obliteration never showing a symptomatology of this kind.

But there are cases in which the diagnosis may be doubtful. Take, for instance, the case of a patient, one of whose lower limbs is colder than the other, the coloration of the skin being red and cyanotic in places. There is no paralysis properly speaking, but the movements of the foot are a little less energetic on the affected side than on the normal side, and there is a state of paresis which renders walking any distance impossible.

The pulsations of the dorsalis pedis and posterior tibial arteries are imperceptible, thus preventing estimation of the blood-pressure by the method of Riva-Rocci and Vaquez. Pachon's oscillatory method does not give precise information either, for it is impossible to distinguish the first increasing oscillation or the last of the large oscillations, when the pointer does not cover more than two or three divisions of the dial in its maximum excursion.

In cases of this kind hesitation is natural, but the problem may be settled by the hot-bath test. The affected limb is plunged into water at 40° C. for ten minutes ; if the case is one of muscular spasm, the pulsations of the dorsalis pedis reappear, and the amplitude of the oscillations increases considerably,

even rising from 1° to 7°, while in the case of arterial obliteration there is no obvious change. This very simple test enables one to distinguish arterial obliteration from vaso-motor spasm, whether of reflex or central origin (Babinski and Heitz) (142).

Late Tetanus and Reflex Contracture

Bérard and Lumière (186), Montais (232), and Courtois-Suffit and Giroux (196) have recently shown that *tetanus* may be manifested by late contractures which develop after the wound has cicatrised (cicatricial tetanus), and become localised and confined more or less closely to the wounded limb (monoplegic form). Such cases as a rule only occur in patients who have had serum treatment. Some of them are not accompanied by trismus at any stage.

Unless care be taken, contractures of this kind may be confounded with reflex or hysterical contractures.

The following is a general account of the characters by which localised tetanic contracture can be recognised—

1. The contracture is *intense:* it cannot be altered by passive movements without causing severe pain, and even then it is difficult to modify it to any extent. "These contractures, which are persistent from the first," write Courtois-Suffit and Giroux, "feel on palpation like a *wooden limb.* As a rule there is a very painful contracture of the whole limb; in Routier's case the thigh was flexed on the pelvis, and the leg on the thigh with the toes strongly flexed, and all the muscles were rigid. In Laval's case the contracture, which had started in the foot and gradually extended to the leg, next day gained the whole limb, which as a result of the generalised contracture was in a position of forced extension, the foot, leg and thigh forming a rigid whole welded to the pelvis like a *bar of iron.*"

2. Extremely painful paroxysmal muscular con-

tractions may be observed resembling the spasm seen in the ordinary form of tetanus, and differing from them only by being confined to the wounded limb. The essential feature of these cramps is that they are provoked by any external cause. The attack begins with a very severe pain, generally starting in the cicatrised wound, and consists in clonic movements which recur at frequent intervals. These localised convulsions are hardly ever absent, being nearly always present at the beginning of the attack, but as a rule they recur at every stage of the disease, and constitute the principal symptom.

3. The contracture of late tetanus is generally not of long duration. It hardly ever lasts more than two or three months, and even that is exceptional.

As a rule there is a slight rise of temperature. The combination of these symptoms is sufficient to exclude the diagnosis of reflex contracture, and of hysterical contracture as well.

Diagnosis of Reflex Paralysis from Hysterical Paralysis

In the first part of the book we described in detail the signs peculiar to hysterical paralysis and contracture on the one hand, and to reflex paralysis and contracture on the other. To avoid needless repetitions we will confine ourselves to giving in tabular form the essential differences between these two classes of nervous disorders.

Reflex contracture and paralysis.	*Hysterical contracture and paralysis.*
1. Paralysis generally *limited* but *profound* and *persistent* even when submitted to methodical treatment.	1. Paralysis generally *extensive*, but *superficial* and *transient* when properly treated.

2. The attitude of the limb in the hypertonic forms *does not correspond to any natural attitude.* The general appearance of certain clinical types is characteristic.	2. The attitudes seen in hysterical contractures usually correspond to *natural attitudes which have become fixed.* Deformities may be observed resembling those characteristic of reflex contractures, but the imitation is imperfect.
3. Amyotrophy usually fairly marked and of rapid development.	3. Amyotrophy is usually absent, even when the paralysis is of long standing, and when present it is poorly developed.
4. *Vaso-motor and thermal disturbances are often very marked,* and are accompanied by an occasionally very pronounced reduction in the amplitude of the oscillations as measured by an oscillometer.	4. There may be thermo-asymmetry, but it is very slight. There are no definite vaso-motor symptoms or modifications in the amplitude of the oscillations.
5. Occasionally very marked hyperidrosis.	5. No definite hyperidrosis.
6. Tendon reflexes often exaggerated.	6. Never any change in the tendon reflexes.
7. Hypotonus sometimes very pronounced, and in paralysis of the upper limb "swinging hand."	7. No hypotonus.
8. Mechanical muscular hyperexcitability often accompanied by slowness of the contraction.	8. No mechanical muscular hyperexcitability.
9. Fibro-tendinous retractions developing rapidly (except in the com-	9. Hysterical paralysis, whether flaccid or with contracture, is generally

pletely flaccid forms, which are rare).	not accompanied by retractions even when it is of long duration. It is even a question whether their appearance does not require the addition of a factor which has nothing to do with hysteria.
10. Trophic disorders of the bones, hair and skin.	10. No trophic disorders.

The recent acquisitions of semeiology thus enable us to make a definite distinction between reflex nervous phenomena and pithiatic disorders.

Hystero-organic, Hystero-reflex and Organo-reflex Associations

Paralyses and contractures due to lesions of the nervous system are distinguished by special objective features, and it is by means of objective features which are absent in hysteria that motor disturbance of a reflex character can be recognised. If each of the various affections which give rise to paralyses and contractures were always to be found by itself, the diagnosis would be relatively easy, but it is far from being so. Morbid associations are, indeed, very common, and tend to complicate the problem.

How is it possible to recognise that there is an association and then to determine the share of each of the constituent elements? It seems to us to be impossible to draw up a formula enabling the question always to be answered correctly. We must confine ourselves to giving a few general indications which may serve as a guide to medical men who are not specialists.

We may remark, in the first place, that to attain the end in view it is essential to know exactly the intensity, extent and evolution of the lesions which may be produced by any given injury of the nervous system,

and to bear in mind what may be called *symptomatic incompatibilities*. But instead of dealing with this question in the abstract we think it best to choose some special examples, and to show in a concrete way the diagnostic criteria of which the clinician can avail himself.

Take a patient who has suffered for three months from hemiplegia which developed suddenly; the paralysis is complete, or almost complete; it is flaccid, and the tendon reflexes are only slightly exaggerated; stimulation of the sole is followed by extension of the great toe. The presence of the last sign indicates a lesion of the central nervous system affecting the pyramidal tract. But it may be asserted at the same time that the motor disorders are only partially due to the organic disturbance, inasmuch as a hemiplegia of sudden onset and of such intensity lasting for three months should be accompanied by a much more pronounced exaggeration of the tendon reflexes and contracture, if it was due to a pyramidal lesion only. This is an example of symptomatic incompatibility which justifies us in concluding that it was a case of hystero-organic association.

Let us take a few examples of various types of associated disorders selected from the neurology of war and relating to patients suffering from paralysis following wounds of the limbs.

A soldier was hit by a piece of shrapnel in the left supraclavicular region, and developed a complete paralysis of the upper limb which was of more than a month's duration at the time of our examination. Electrical exploration showed a very marked reaction of degeneration in the muscles innervated by the musculo-cutaneous nerve, and diminution of electrical excitability in the muscles innervated by the musculo-spiral; on the other hand, the electrical excitability was normal in the muscles supplied by the circumflex, ulnar and median. There were no vaso-motor symptoms. These findings suggested an *hystero-organic association*, and we were immediately able to confirm

this hypothesis. We declared that electrical treatment was bound to bring about an almost complete cure, and in conformity with this assertion the patient, after being subjected for a few months to induced currents, was able to work all the muscles of which the faradic contractility was normal; thus he was able to raise the arm, flex the wrist and fingers, close the hand, and extend the hand and fingers. Flexion of the forearm remained the only difficult movement, which was easily explained by the reaction of degeneration in the muscles on the anterior aspect of the upper arm; the partial execution of this movement was also explicable, and was due to the action of the supinator longus.

Another patient after a wound in the axilla developed paralysis with R.D. of the circumflex, musculo-cutaneous and musculo-spiral; he was also unable to carry out any movement of flexion of the hand and fingers, and yet the electrical excitability of the muscles on the anterior aspect of the forearm was normal at the time of examination, *i..e.*, three months after the onset of the symptoms; there were no vaso-motor troubles. There was every reason for supposing that this paralysis was entirely psychical, and as a matter of fact it was possible to make it disappear in a few moments by purely psychotherapeutical methods.

In other cases, the same muscular group may be at once the seat of an organic paralysis due to a lesion of the nerves, and a psychical paralysis grafted on the latter and aggravating the disturbance of function.

Lastly, the hysterical paralysis may be associated with the organic paralysis in an exceptional manner, the organic symptoms, as in the cases which we describe below, being preceded by hysterical symptoms (*v.* p. 624). The term *organo-hysterical association* was recently proposed by one of us to distinguish cases of this kind from the ordinary type of association (68).

A patient wounded in the upper part of the forearm showed the following symptoms : in the first place, in the distribution of the ulnar nerve, a paralysis with

reaction of degeneration of the hypothenar muscles, adduction of the thumb and interossei, and, secondly, an incomplete paralysis of the muscles of the hand innervated by the median and a paresis of the extensors and flexors of the fingers. These last muscles did not show R.D., but their mechanical excitability was exaggerated and the contraction caused by percussion was slow. Moreover, the whole of the hand, irrespective of the nervous territory involved, was cyanosed and cold. This was an example of an organo-reflex association and not of an hystero-organic one.

We will now take a few examples relating to the association of reflex motor disturbances with hysterical or pithiatic phenomena (*hystero-reflex association*).

As a result of a wound in the left leg a soldier presented a remarkable gait, which constituted a form of rhythmic chorea; at every step he bent his knees as if he were going to sit down, and then he raised himself again. But he also showed vaso-motor and thermal disturbance of the left lower limb, mechanical hyper-excitability of the small muscles of the foot and loss of the plantar cutaneous reflex with its transitory re-appearance under the action of heat. Such symptoms can hardly be explained except by the association of hysterical manifestations with reflex nervous disorders, and, as a matter of fact, counter-suggestion rapidly cured the disturbance of gait which was due to hysteria, but it had no. action on the vaso-motor or thermal troubles, nor on other symptoms of the same kind observed in the wounded limb.

In the cases which we have just described the clinical problem was extremely simple, as it was very easy to distinguish the influence of each of the two factors in this association.

Another soldier developed complete brachial monoplegia after the lower part of the forearm had been perforated by a projectile without, however, causing any lesion of the nerves or vessels. All the movements of the different segments of the limb were abolished. The hand and forearm were slightly atrophied and

presented a salmon-red coloration. The temperature of these areas was 3°–4° C. lower on the affected side than on the sound side. The oscillations measured in the forearm with Pachon's sphygmometer were half the size of those on the sound side, but the systolic pressure (Riva-Rocci-Vaquez) was normal. There was mechanical muscular hyperexcitability and slight exaggeration of the osso-tendinous reflexes. There is no doubt that this paralysis is to some extent of a reflex character; but, inasmuch as hitherto no case of complete monoplegia has been found in definite cases of reflex disorders, but merely limited paralysis in which the extremity of the limb only is considerably affected, there are grounds for maintaining that hysteria enters to a large extent into the causation, and that we are dealing with a hystero-reflex association.

The same applies to another patient who is suffering from complete paraplegia of the legs following frostbite. Both his feet show vaso-motor symptoms, hypothermia and mechanical hyperexcitability, which are characters common to reflex phenomena. But these disturbances, intense as they are, appear incapable of producing a complete paraplegia of the legs. The paraplegia in question, not being accompanied by any of the objective signs indicating lesion of the nervous system, must be attributed to hysteria.

It seems unnecessary to give any more examples. We will merely remark that the modes of association may be even more complex than in the cases which we have quoted. The nervous symptoms found in a patient are sometimes derived from three sources: a nervous lesion, reflex action and hysteria (hystero-reflex-organic association), and even that is not all.

The preceding disorders may be complicated by disturbances of the functions of the wounded limb resulting from changes in the muscles, tendons, bones and vessels, as well as from cicatricial adhesions and retractions, which are so common after the wounds of warfare.

It is chiefly owing to the neglect of morbid associa-

tions that confusions and errors of interpretation were formerly, and are still, committed. It was on this account that suggestion carried out in hypnotic sleep or in the waking state was recently accredited with the faculty of attenuating and even curing organic affections (*v.* pp.415 *et seq.*). The same reasons led to such symptoms as exaggeration or loss of the tendon reflexes being classed with the disturbances produced by hysteria.

Let us consider one of the cases of hystero-reflex association which we have already described. Counter-suggestion at once cures most of the motor disturbances, and the exercises performed by the patient warm the limb, which was previously in a condition of hypothermia. The success of this treatment might give rise to the hasty conclusion that the vaso-motor and thermal symptoms were hysterical in origin. But, if one follows up the patient, it will be found that conclusions drawn from the therapeutic test were premature and unjustified. The vaso-motor symptoms persist, the hypothermia returns, the disappearance of the motor disturbance is not complete, and the more or less considerable residue which often causes great discomfort as well as inaptitude for military service is refractory to counter-suggestion, and can only be improved slowly and progressively by physical methods and not by psychotherapy.

Hysteria and Simulation

Works dealing with simulation prior to Chavigny's *Diagnosis of Simulated Diseases* (193), did not attack the question of hysteria. Certain methods, therefore, of diagnosis advocated in these earlier text-books, and founded on surprise or violence, should be rejected. "If there were only diseases with a palpable and anatomical substratum," Chavigny very properly remarks, "these methods would have a real value, but it is just the peculiarity of hysteria and some neuroses

resembling it that the symptoms can be modified at will by these methods of surprise, violence or trickery." "The most characteristic feature of hysteria," he adds, "is the variability of its symptoms and their essentially functional character without an anatomical basis. It is precisely this variability of symptoms which makes hysteria difficult to distinguish from simulated disorders, which share the feature of symptomatic inconsistency. . . . Simulation is not necessarily proved by catching the patient tripping."

It is unnecessary to follow the writer in his examination of various hysterical symptoms such as stigmata, anæsthesia, paralysis and contracture. Although he unhesitatingly adopts the old conception of hysteria, he shows how variable and unstable these symptoms are, and how liable they are to be taken for mere fraud. These remarks so closely resemble the ideas which we have expressed that we need not dwell any further on the matter, but will merely remind the reader of what one of us wrote: "The hysterical subject always behaves as if he were partly able to control his disease, and as if his sincerity was not absolute; contrary to the epileptic he hardly ever has a fit, except under certain circumstances, and almost always recovers from the attack without being injured by the convulsions which frightened the bystanders; though a prey to terrifying hallucinations, he does not, like the alcoholic in his hallucinations, commit acts which are dangerous to himself; though he has a very deep thermal anæsthesia, he will not expose himself to the risk of being burnt like the syringomyelic, and a narrowing of the visual field, however pronounced it is, will not prevent him walking about and avoiding obstacles, as it would if the visual constriction were organic. All this makes hysteria resemble simulation, and I am in the habit of saying that the hysterical subject is to a certain extent a semi-simulator" (*v.* p.431).

Does this mean that a distinction between a phenomenon due to simulation and one due to hysteria is

impossible? Chavigny does not hold this opinion. He thinks that hysteria may be recognised by the following characters—

1. Special disturbance of the cutaneous and tendon reflexes, loss of the cutaneous reflexes and dissociation of the reflexes consisting in an abolition of the plantar reflex with exaggeration of the knee jerk.

2. Fixed condition of the hysterical contracture (absence of variation in the patient's resistance when an attempt is made to overcome the muscular rigidity).

3. Special forms of anæsthesia, the simulation of which would be impossible, and the presence of permanent stigmata.

There is no need to make a fresh criticism of these so-called distinctive characters. We have shown that hysteria is incapable of modifying the condition of the cutaneous and tendon reflexes (*v.* pp. 416 and 471), and that hysterical contracture does not differ objectively from a voluntary contracture (*v.* p. 463). As regards the anæsthesia, although it is difficult to simulate this symptom when it is due to an organic cause owing to the special features which it then assumes (permanence, definite distribution, etc.), it is not clear why hysterical anæsthesia should be difficult to simulate, since it is essentially variable and unstable, as Chavigny himself recognises. What is there to prevent the conscious will carrying out what is readily done by auto- or hetero-suggestion?

Simulation can reproduce everything that hysteria or pithiatism is capable of doing. Anything that is impossible for simulation is also impossible for hysteria. This observation ought not, however, to lead us to confound hysteria and simulation. Some time ago, one of us, in pointing out the difficulty of this distinction, expressed himself as follows, in a lecture in which he set forth his views on hysteria and hypnotism (34)—

"As a careful examination of the facts leads to eliminating from hysteria all the objective phenomena

which suggestion is unable to produce, some of you may perhaps be inclined to wonder if hysteria is really a pathological condition, and if so-called hysterical manifestations are not merely due to simulation. The following is my opinion on this question, which deserves careful consideration. From the observation of very many hysterical patients whom I have followed up I have derived the conviction, which is shared by every neurologist, that many of these patients are sincere and cannot be regarded as simulators, but I must confess that this idea is founded on moral considerations and cannot be proved with the scientific rigour employed in the study of organic affections. A clever and well-trained simulator may succeed in closely reproducing every hysterical symptom, which is a source of almost insurmountable difficulty in medico-legal reports relating to cases of hystero-traumatism.

"How is it possible, then, to distinguish the true from the false? This is a difficult problem to solve. It must be confessed that it is impossible in any particular case to determine with certainty the degree of the patient's sincerity; circumstances alone enable one to form a probable opinion on this point. There is every reason for supposing that the unobtrusive symptoms of which the patient does not make any display are most probably genuine; this is so in particular in associated hysteria in which pithiatic phenomena become insidiously grafted on organic symptoms without the patient appearing to be conscious of their mechanism."

This view has been confirmed by the cases observed in the neurology of war. We will mention here an example of organo-hysterical association (68) which occurred under the following circumstances. The case was that of a lieutenant who developed an hysterical crural monoplegia after a contusion of the right thigh. Although the paralysis was of several months' duration, the tendon and cutaneous reflexes and electrical excitability of the muscles were absolutely normal, and the result of psychotherapy confirmed the

diagnosis of hysteria. But there was also a right musculo-spiral paralysis which was obviously organic, due to compression of the nerve by the crutch used by the patient. The following remarks accompanied the description of the case : " This association in my opinion is a remarkable one, because it seems to me to prove that it would be an error to confound hysteria and simulation, as is the tendency with some writers nowadays. I have myself insisted, it is true, on the difficulty of separating simulated phenomena from those which are the result of suggestion, and on the absence of objective characters enabling a line of demarcation to be made between these two conditions. I have even said that hysteria is a sort of semi-simulation, but this expression clearly indicates that the simulation is not complete. In the present case it may be affirmed that the patient was sincere and thought that he was quite incapable of moving his leg or standing on it, since with the object of remedying this imaginary loss of power he had been accustomed to support himself so conscientiously on his crutch that an organic paralysis had resulted. I may add that the patient did not inform me of this musculo-spiral paralysis except as an afterthought. It may be said, therefore, that a purely imaginary disorder occupied a much more important place in the patient's mind than the real disorder which accompanied it." The hypothesis of simulation could also be dismissed in another case, reported by Ivan Moricand (80), in which bilateral musculo-spiral paralysis from compression developed in a soldier suffering from hysterical crural monoplegia as the result of the prolonged use of crutches.

Other arguments might be given in favour of the sincerity of patients suffering from hysteria or hystero-traumatism, such as the joy and sincere and lasting gratitude expressed by the patient after his recovery, as well as the devotion to duty and sometimes even actual heroism shown by the man who returns to the firing-line. The value of these arguments is not to be

underestimated, although they are exclusively of a moral character.

The question of simulation has been dealt with since the beginning of the war at the Neurological Society (91).

In the first place, Gilbert Ballet proposed the following definition in order to give a precise meaning to the terms employed in the course of the discussion— "Simulation: a subjective or objective disorder invented by the patient with the object of voluntarily and consciously misleading the observer. Closely connected with it is the conscious and intentional exaggeration or prolongation of a real disorder."

As regards simulation most neurologists are agreed that there are two absolutely certain criteria: being caught in the act, and confession. But it must be admitted that confession is not conclusive in every case, as it may be nullified by the methods employed to elicit it, and the mental state of the individual. Moreover, magistrates are agreed that confessions are only of value if corroborated by the actual facts.

As regards being caught in the act, this, of course, cannot apply to the observation of a sudden movement in a case of contracture or paralysis. The term "caught in the act" can really only be used when the patient who pretends to be suffering from a nervous disorder, *e. g.*, deafness or a motor disability, constantly behaves like a normal person as soon as he thinks that he is out of the range of medical observation.

Cases of genuine simulation appear to be very rare. Although there are others in which simulation may be suspected, it must be laid down that, in the absence of decisive proofs, the subject under suspicion should benefit from the doubt. Not only should the diagnosis of simulation not be made, but the mere word "simulation" should not be uttered in such cases, considering the gravity of the accusation under present circumstances. There is no practical objection to this method of procedure, which is the only fair one. One may, indeed, follow the same line of conduct in every case

of exaggeration or simulation as in hysteria, and have recourse to psychotherapy in its different forms, just as if it were a case of pithiatism. Judging by our experience, these disorders of doubtful nature are as a rule cured in this way, provided that energy, tact and perseverance be employed.

CHAPTER X.

TREATMENT AND DECISIONS BY MEDICAL BOARDS

The treatment required for pithiatic manifestations differs considerably from that needed in cases of reflex contracture and paralysis. Decisions by medical boards cannot be the same in the two conditions. We have thought it advisable, however, to consider them together, for their frequent association often renders necessary a simultaneous employment of psychotherapeutic and physiotherapeutic methods. We shall thus be able to illustrate for the last time the fundamental differences between them.

Treatment of Hysteria

As in most other affections, the medical man should have two equally important aims: first, to prevent the development of pithiatic disorders (hygiene and prophylaxis), and secondly to cure them when once they have developed (therapeutics).

Hygiene and prophylaxis.—The determining cause of hysterical manifestations is auto-suggestion and hetero-suggestion. The patient sometimes derives the first idea and the theme of his disorders from his inner self (auto-suggestion), his present or past sensations, his experience, the true or false notions which he has acquired, and from his meditations. Various pithiatic disorders may thus arise a few days or even a few hours after a physical or moral shock, without it having been possible to use any method to prevent their development.

But they often appear much later, *e. g.*, in the hospital

to which the patient has been removed. The patient's removal may have been due not to a neuropathic state, but to a contusion or wound sometimes of a trifling nature.

Auto-suggestion is also liable to intervene in such cases, but it is almost always combined with hetero-suggestion, which then usually constitutes the essential factor.

This hetero-suggestion is derived from the entourage, the friends, and all who come near or look after the wounded man. Nurses, with the best possible intentions, often exercise a very bad influence on their patients by their questions, attitude and injudicious attentions. Their emotion, anxiety, compassion and continual desire to save the patient the slightest effort or discomfort, all constitute the most powerful agents of suggestion.

Doctors themselves, who do not sufficiently realise the good or bad effects which their statements, comments, examinations and gestures may have upon the mind of their patients, whom suffering, privations and moral or physical commotions have made very responsive to suggestion, doctors, we say, sometimes unconsciously exercise a hetero-suggestion which is all the more powerful as their profession gives them authority. They should always be alive to this possibility, and should place their patients under the least favourable conditions for suggestion.

They must keep the wounded away from demoralising influences, set an example to their staff, supervise the entourage and keep a perpetual watch upon themselves. Every one knows of the ward epidemics which were so common in certain hospitals in former times. In those days it was not uncommon to see during the doctor's visit several patients in hysterical convulsions simultaneously or successively. Although these manifestations of collective hysteria have disappeared, thanks to the development of ideas and their practical results, a reappearance of such epidemics might be witnessed if the conditions were suitable for their development.

Convinced of the rôle played by suggestion in its various forms, neurologists, at a meeting devoted to the study of so-called functional disorders, were all agreed as to the dangers involved by writing on the soldiers' evacuation cards such diagnosis as "epileptic attacks," "epileptiform attacks," "sciatica," "paralysis," and "cerebral" or "spinal commotion." This card, which the soldier keeps in his book and may constantly be reading again and again, gives rise to the most harmful reflections. All the vague apprehensions to which the patient is liable are made concrete by this diagnosis. It gives a genuine character to his complaints, a most regrettable occurrence, as may be imagined, when an error of diagnosis has been committed and he is suffering from hysteria.

According to the recommendation made at the meeting, medical officers dealing with the evacuation of cases in order to avoid any suggestion should confine themselves to giving a detailed account of the symptoms which they have found, and refrain from recording their diagnosis, which should only be done after a prolonged examination in special centres.

It is obvious therefore, that there is what may properly be called a hygiene and prophylaxis of hysteria, and it is easy to understand its importance under present conditions.

Therapeutics.—What is the right way to treat pithiatic disorders, once they have developed? As every one knows, recourse should be made to psychotherapy, persuasion and counter-suggestion. A priori, this appears very simple. It might be supposed that it is enough to tell the patient that his affection is a purely imaginary one, and ought to disappear by an effort of the will. But, in reality, a formula of this kind is too simple. In order to be successful, persuasion and counter-suggestion require the presence of various conditions which we will now discuss.

An all-important condition is the creation of an atmosphere of *confidence and faith.* It may be said generally that confidence in the doctor is beneficial to

the patient, but this is particularly true as regards the hysterical patient. He has already taken a step in the right direction when he has gained the conviction that his doctor has the power to cure his troubles. He readily acquires this conviction when he has seen rapid recoveries from what he thought were severe attacks in the cases of other patients, or when his ward companions tell him that they have been cured by methods similar to those which are proposed for him. His confidence increases in proportion to the frequency of the recoveries and the number of favourable reports which he hears.

The environment, therefore, exercises an influence which, instead of being harmful, as it so frequently is, constitutes a most valuable help. But an environment of this kind which is in sympathy with the doctor and beneficial to the patient is not made to order, but gradually develops as the result of successful treatment which strikes the attention and provokes the astonishment and admiration of the bystanders, especially if it is immediate. The first successes are the most difficult to obtain; as they increase in number there is less difficulty with them. In the first trial of psychotherapy it is essential not to spare time or trouble, but to attain the end at all costs. It goes without saying that an accurate diagnosis is indispensable for success.

To treat an organic or physiopathic affection by psychotherapy, and to promise a rapid recovery in the case of a patient suffering from a non-pithiatic disorder, *e. g.*, a reflex contracture without any association with hysteria, and in such a case to employ counter-suggestion of any kind, is not only to waste time, but, what is more important, to lose prestige and to bring discredit on the method.

The disappearance of the symptoms should not be promised until a complete examination has been made and the diagnosis of hysteria has been firmly established. In a case of associated hysteria the patient should be told that his trouble will improve, but he

should be given to understand that the improvement will only be partial. One should then set to work and persist if necessary till success has been attained.

It is in this way that the neurologist creates for himself, to the great benefit of his patient, the reputation of being infallible in his cures. His psychotherapeutic power becomes very great as soon as there is no doubt as to the certainty of his diagnosis, and the accuracy of his prognosis and statements generally.

Cure is, as a rule, easily obtained when an *early treatment* has been started, as can readily be understood. Under opposite circumstances there is more difficulty: the older the disease, the more deep-rooted it is in the mind, and the more automatism intervenes to keep it there. In hysterical contracture or tremor, for instance, all the patient's attention is at first required to keep up the disorder, but later he holds his arm stiff or swings it about automatically almost without thinking about it, as in the act of walking. Charcot said many years ago: "Contractures should not be allowed to become chronic"; the same applies to all hysterical manifestations. This justifies the conclusion that hysterical soldiers should be treated as far as possible in *units at the front.* Medical officers who have done this systematically have obtained excellent results. It should not, however, be inferred from these remarks that hysterical symptoms of long duration are incurable, but more perseverance and energy are required to cure them.

Whether we are dealing with a recent or an old-standing complaint, it is important in our opinion to have recourse to what may be called the *abrupt treatment*, which we consider to be far superior to slow and progressive psychotherapy combined with isolation.

It may be laid down as a rule that it is very desirable to obtain a definite improvement at least on the first application of treatment. The first interview or encounter with the patient presents particularly favourable conditions from the therapeutical point of

view. It is at this moment that the patient's faith in his doctor's power is usually most active, and with this sentiment is associated a sort of apprehension, emotion and anxiety which favours the curative process. We have shown that deep emotion is incompatible with hysterical manifestations. It is not the same with an emotion of a lesser degree; it may, however, result in distracting the patient's attention from the object of his suggestion. The person under the influence of suggestion should be regarded in the light of an actor on the stage (but not as a simulator, as we have already explained). A moral shock, a fright or a mere surprise caused by an unexpected occurrence may change the course of his ideas, and make him forget his part for the time being. Thus a patient with hysterical paralysis, owing to the emotion and bewilderment caused by the doctor's numerous questions, orders, tests and mysterious medical rites, will for the moment forget his part, and carry out a few movements with his paralysed arm or leg (*v.* p. 591). This is the moment which the doctor must be on the look-out for; the patient should at once be shown that he is no longer a cripple. In his state of confusion and stupefaction he will be persuaded to reproduce the movements which occurred almost unconsciously before. It is most important then to persevere and to try to obtain at once a complete cure, or at least a definite result in conformity with the doctor's forecast and promises.

The method of treatment should vary somewhat with the mentality of the subject, his intellectual and social level, degree of education, state of mind and desire to recover. A cultivated man should be given to understand that the art of diagnosis has now attained such a degree of perfection that there is complete justification in his case for excluding the idea of any organic lesion. He should be told that his condition has been caused by suggestion and is undoubtedly neuropathic, and that he will be sure to recover. If such statements appear to humiliate him he should

be persuaded that such a state of mind, after the shocks which he has experienced, is quite compatible with normal intelligence, an excellent moral and great courage. Persuasion alone without any physiotherapeutic assistance may then produce an immediate and decisive effect. It is in cases of this kind, it may be said in passing, that the diagnosis of hysteria can be undoubtedly established.

But in dealing with patients who are less able to follow a scientific train of thought, it is advisable to strike their imagination by employing a material method as well. Electrical treatment is one of the commonest and most successful methods, as can readily be understood. The laity, in fact, readily believe in the power of electricity; moreover, the movements caused in a paralysed leg by variations in the intensity of the current are calculated to impress the imagination and to dispel the idea of a loss of strength; lastly, the physical and moral impression caused by electricity is well calculated to produce the distraction and surprise of which we have endeavoured to show the good results.

It may be advisable for the same reason to have recourse to various exercises likely to bring physiological automatism into play. For instance, what should be done in the case of a paraplegic patient with contracture, who, in spite of every variety of treatment, appears incapable when in bed of carrying out the slightest voluntary movement?

With the help of an assistant the doctor should pull him suddenly out of bed, put him on his feet, and drag him forwards, taking care to prevent his falling. Very often in such cases the patient, taken unawares, takes a few steps to preserve his balance. As in the case quoted above, his attention should at once be drawn to this result; he should be told that his recovery is imminent, and the movement should be accelerated without leaving him a moment's rest, the fatigue which he feels constituting a favourable circumstance.

Meanwhile, you must keep on encouraging him in

every way, and as soon as you feel that he has almost gained his balance, which usually occurs if you have the patience to wait, you may leave him to himself, taking care to prevent him from falling, as you would with a child learning to walk. The conviction thus acquired, that his paralysis is no longer complete, constitutes a success. Complete recovery henceforward takes place without much difficulty and may be rapid —the patient, who has been brought on a stretcher, sometimes walking back to the ward by himself after the first treatment.

But such results, it must be said, usually require a prolonged effort on the part of the doctor, who must not spare either time or trouble. The patient, who was sceptical and recalcitrant at first, gradually begins to show confidence and ends by helping the doctor as much as he can. On taking leave he expresses his joy and gratitude with obvious sincerity—a scene which we have often witnessed.

At the beginning of the war neurologists were not all agreed as to the results to be obtained, especially in hysteria of old standing. Some of them regarded the condition as permanent and refractory to any method of treatment. Some even were in favour of a discharge from the army. This view has been discussed, but fortunately it has not been generally accepted. The great majority of neurologists rightly considered that such a procedure would be regrettable in every respect, and at its meeting on October 21, 1915, the Neurological Society sent a recommendation to the Under Secretary of State for the Sanitary Service to the effect that "*no soldier at the present time, under any circumstances, with a psycho-neurosis should be brought before a medical board with a view to discharge from the army.*"

Personally, we have long insisted on the possibility of an almost certain cure of purely hysterical manifestations (*v.* p.421), and this opinion has been strengthened by what we have seen in the course of the last two years. In dealing with recent disorders our task is

relatively easy. If they are of old standing they sometimes offer great resistance to treatment; however, even then they end by giving way, if the conditions which we have specified are realised. This is the result of observations made by several of our colleagues, especially Souques, Meige, Albert Charpentier, Clovis Vincent, Roussy and Léri, and is also the outcome of the numerous observations which we have made ourselves since the beginning of the war. Clovis Vincent, who had the opportunity of treating a very large number of hysterical cases, informed us that he had cured almost all of them.

There is no need to add that in cases of association the hysterical disorders are the only ones to disappear under the influence of the methods such as we have described. Disorders of an organic or physiopathic nature associated with the preceding persist, and are not likely to be affected except by other modes of treatment. In the neurology of war these associations are particularly frequent, which doubtless explains why, after a considerable improvement in disorders treated by psychotherapy, more or less obstinate and important sequelæ often persist.

Treatment of Reflex Nervous Disorders

We have just remarked that hysteria was fairly frequently associated with other affections; among these reflex nervous disorders take a prominent place. This is one of the reasons why the latter have for a long time been confused with hysterical manifestations.

The frequency of this mode of association, in which it is not always easy to distinguish the part played by each of the constituent elements, is an encouragement to make an almost invariable use of *psychotherapy* in undoubted cases of reflex nervous disorders. No harm can result from this, provided that the rules mentioned above be strictly complied with, so as not to bring discredit on the method; but it cannot be too frequently repeated that counter-suggestion has

no action on the reflex troubles themselves. It is, therefore, advisable to adopt *physiotherapeutic methods* at once as well as the measures already mentioned.

This method of treatment, however, frequently involves a hidden danger which it is important to avoid. Violent exercise, intensive mechanotherapy and electrotherapy, and massage accompanied by energetic kneading of the muscles, have often been followed by an aggravation of the contractures, as a large number of observers have found. The use of these methods is, however, justifiable, provided they are undertaken with gentleness and circumspection and under medical supervision. We would go so far as to say that recourse should be made as soon as possible to *continuous extension* and *gentle and methodic movement*, to avoid, as far as possible, the formation of fibrous retractions which takes place so early in such cases.

Considering the importance of the vaso-motor symptoms and the hypothermia, it appeared natural to make a trial of *thermotherapy* in its different forms (balneotherapy, hot-air douches, and light baths). Diathermia, judging from our experience, seemed very beneficial. It was followed in many cases by progressive improvement, and we could hardly believe that this was a mere coincidence. There is no need to add that great prudence is still required, as high temperatures are not well borne, and are likely to do much harm. The results obtained were not always the same, and in our experience were never rapid.

In view of the chronic character of these disorders and the mechanical obstacles created by fibrous retractions, it was necessary to have recourse to surgery. Sudden correction under anæsthesia, and tenotomy followed by immobilisation in splints, had decidedly unfavourable results Sicard appeared to be pleased with the use of alcohol injected into the nerves in some cases of obstinate acro-contracture.

In flexion contractures of the leg on the thigh L. Bérard successfully performed continuous extension combined with injections of novocain (1 per cent.

solution) into the trunk of the sciatic nerve and the contractured muscles.

The presence of scars adherent to the deep parts seen in cases of this kind justifies operative interference. Tixier obtained encouraging results by *excision of the scar and dissection and liberation of the subjacent tissue;* one of his cases was published by Duvernay (124).

Leriche advocated the tearing apart of the periarterial plexuses in causalgia (130) (143), and performed the operation in cases of reflex contracture with pronounced and obstinate vascular spasm with interesting results. He has written to us about the operation as follows: "I operated on three cases of reflex contracture of the upper limb in which I excised the plexuses of the brachial artery, and all three showed distinct improvement. One patient, as the result of the operation, was able to extend his contractured fingers in which the nails were causing ulceration of the skin; as soon as this result had been obtained, it was possible to practise continuous extension and re-education of movements, which had hitherto been impossible. In another patient, who had an 'accoucheur's hand' contracture following a through-and-through wound of the wrist, the operation was followed in a few days by considerable improvement in the movements of the fingers, which became easier and more extensive. The improvement coincided with an increase in the blood-pressure in the limb which had been operated on (Heitz), and a very decided rise in the local temperature."

But it must be admitted that these questions are not yet definitely settled, and that we must wait before giving a final opinion as to the respective value of these different methods.

As regards the question of decisions by medical boards on wounded soldiers who show nervous disorders of this kind, we will simply remind our readers of the conclusions drawn up by one of us, and unanimously adopted at the Neurological Society at the end

of a discussion devoted to the study of so-called functional nervous disorders (140)—

" Regarding the matter exclusively from the standpoint of a medical board we have to distinguish among so-called functional motor disorders (*i. e.*, motor disorders with none of the objective signs characteristic of organic affections of the brain, cord or nerves, or of vascular lesions)—

" 1. Hysterical or pithiatic disorders properly so called.

" 2. Nervous disorders quite distinct from the preceding, associated with real physiological disturbances, the mechanism of which is still a matter of discussion, but which may be grouped with the reflex disorders observed after osteo-articular lesions.

" The nervous symptoms belonging to the second group appear in the form of paralyses or contractures which are more or less complete and assume different types. They are accompanied by the following objective signs which enable them to be distinguished from the nervous disorders of the first group, viz. pronounced and obstinate vaso-motor and thermal troubles, modifications of muscular tonus (hypotonus, hypertonus, and association of hypotonus and hypertonus), increase in the mechanical excitability of the muscles and sometimes even of the nerves, quantitative changes in the electrical excitability of muscles without R.D., muscular atrophy and trophic changes in the osteo-articular tissues and sometimes in the skin. Contractures belonging to this group are characterised by their prolonged resistance to Esmarch's band and general anæsthesia. Although there is no reason for denying that the disorders in this group are curable, it must be recognised that even under the most favourable circumstances they are sometimes very persistent.

" Before any decision is made by a medical board, it is important that wounded men suffering from this variety of motor disturbance should undergo a prolonged examination at a neurological centre, and that every appropriate psychotherapeutic and physio-

therapeutic method should be employed simultaneously. But after all attempts at treatment have proved a failure, it does not appear advisable to prolong the stay in hospital indefinitely in cases where the reality of the physiological disorders mentioned above has been established beyond a doubt. Leave for convalescence may be granted, but on condition that the patients on the expiration of their leave are sent back for examination or fresh treatment to the same neurological centre. In obstinate cases discharge from the army may be suggested, but this should always be a temporary one."

At a recent meeting of the representatives of the neurological centres (December 15, 1916) to discuss the subject of " Discharges from the Army, Disabilities and Allowances in the Neuroses," the questions with which we have dealt in this book were discussed from the point of view of a medical board, and the conclusions of a report drawn up by one of us were adopted to serve as a groundwork for the next edition of *The Ready Reckoner of Disabilities*.[1]

They are of interest to all medical officers, and may therefore be reproduced here. They are as follows—

1. For purely hysterical or pithiatic disorders : no discharge nor allowances.

2. In cases where pithiatic symptoms are associated with organic, physiopathic or mental disorders, the hysterical manifestations should not be taken into account in estimating the degree of disability.

3. In the physiopathic or so-called reflex nervous disorders following wounds in war and refractory to prolonged treatment, service with the auxiliary forces or temporary discharge with allowance proportionate to the loss of function is indicated.[2]

[1] *Guide-Barême des Invalidités.* Published by Lavauzelle.

[2] It should be understood that the degree of disability in the case of reflex paralyses and contractures should be calculated by *The Ready Reckoner of Disabilities* according to the scale

It is clear from these recommendations and from the discussion which took place at the meeting, that there is no doubt as to the distinction between hysterical or pithiatic disorders and the so-called reflex disorders. It is well recognised that the prognosis, treatment and administrative measures differ entirely in the two conditions. It is generally agreed that they should be separated and that so-called reflex nervous disorders are more closely allied to organic disease.

The possibility of granting a temporary discharge and allowance to patients suffering from reflex disorders constitutes an equitable measure. It would be unjust and prejudicial to the general interest to keep patients suffering from physiopathic disorders on which the will has no effect, indefinitely in isolation wards, as has been done sometimes on the suspicion of aboulia and even of simulation.

In their recommendation that there should be no discharge or allowance in purely hysterical disorders, the representatives of the neurological centres proved that they shared the ideas which have been set forth in this book, and have long been maintained by one of us.

Neurologists to-day are almost unanimously agreed that hysterical or pithiatic disorders should be almost infallibly cured by persuasion or counter-suggestion when the conditions required for the treatment are realised. It would, indeed, be difficult to dispute this, considering what remarkable results have been obtained in neurological centres at the front and at the base, where the number of recoveries from hysterical or pithiatic disorders has been almost incalculable.

It is no exaggeration to say that an extremely

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used for determining the degree of disability in organic nervous diseases. The specialist's opinion should be drawn up without the patient's knowledge, and sent off in a sealed envelope to prevent its giving rise to claims and counter-claims. Although the medical expert should pay careful attention to it, it must not be forgotten that he is the only one legally qualified to fix the amount of the allowance.

useful service has thus been performed. Thanks are due to the Neurological Society, which from the first year of the war has clearly specified what line of conduct should be followed in hysterical cases, and has demanded the creation of special services; to the heads of the different centres and their assistants, whose efforts have been incessant; and, lastly, to the Under Secretary of State for the Sanitary Service of the Army, for having supported all these efforts, encouraged initiation, and completed the organisation of neurological centres.

POSTSCRIPT

After the manuscript of the French edition had gone to press some new publications appeared, the conclusions of which are in more or less complete disagreement with our own. Some discussion of them therefore appears necessary.

Generally speaking, this is the line of argument adopted by our opponents. They maintain that there are no physiopathic contractures or paralyses of a reflex character; the motor disorders considered as such are pithiatic phenomena; as regards the amyotrophy, circulatory disturbances, cyanosis, hypothermia, mechanical hyperexcitability of muscles and nerves with slowness of the muscular contraction and premature fusion of the faradic contractions, hypotonus, and exaggeration of the tendon reflexes, all these are merely disorders due to immobilisation or inactivity in association with pithiatic motor disturbances.

The principal argument brought forward in support of this view consists in the therapeutical results said to have been obtained, such as the cure of motor disorders by psychotherapy and progressive improvement in other symptoms under the influence of methodical exercises. It should be noted that this second part of the treatment requires a relatively long time, even in the opinion of its most enthusiastic advocates. Roussy, Boisseau and d'Oelsnitz,[1] for instance, think

[1] Roussy, Boisseau et d'Oelsnitz, La station neurologique de Salins (Jura) (centre de psychonévroses) après 3 mois de fonctionnement. Rapport présenté à la Conférence interalliée pour l'Etude de la Rééducation professionnelle (Paris, 8–13 Mai 1917) et publié dans le *Bulletin de la Réunion Médico-Chirurgical de la 7e Région*, Besançon, Juin 1917.

that an average period of three to four months is necessary to obtain a cure.

Without disputing the excellence of the results obtained by a combination of psychical and physical methods, under particularly favourable conditions, we are guarded as to the general possibility of obtaining a complete cure of physiopathic disorders within a definite period when they are very pronounced. We mean by complete cure a return to the normal condition identical with that in which the patients were before the traumatism.

We may admit provisionally that most of the patients belonging to the class in question recover after a treatment of three to four months under the influence of psychical and physical treatment combined, if carried out under the best conditions. Must we conclude from this that physiopathic symptoms are merely the consequence of pithiatic motor disorders, and are due to immobilisation produced by hysterical paralysis or contracture, and that the so-called reflex nervous disorders should be regarded as hysterical? We do not think that such a conclusion is justified, and we will state some of the reasons on which our opinion is founded.

It is very common to find hysterical contractures and paralyses of very long duration, *e. g.*, two years and even more, unaccompanied by any physiopathic disorder, such as amyotrophy, vaso-motor symptoms or hypotonus. It is therefore difficult to suppose that immobilisation of this kind which has not caused any physiopathic disorder in some cases after lasting for so long, may cause very marked disturbances in others after a relatively short lapse of time. Is it not logical to suppose that another factor must have intervened?

But, if hysterical immobilisation which the patient has accepted of his own free will, so to speak, is insufficient, is it not possible that these disorders may be produced by the immobilisation due to organic disorders or wounds causing pain and necessitating the application of a splint? This kind of immobilisa-

tion, which the patient has not desired but to which he has had to submit, is no doubt more injurious, but it does not appear to be an adequate explanation of all these physiopathic phenomena.

We showed the resemblance of these cases to those observed by Charcot and Vulpian after osteo-articular lesions, and described by them under the names of reflex amyotrophic paralysis and reflex contracture, and invoked a mechanism of the same kind to explain their origin, the expression "nervous disorders of a reflex character" merely implying the idea of a disturbance of the nerve centres.

This was merely an interpretation which we put forward, and we remarked that its non-acceptance would not in any way modify the question at issue. Our object was to draw attention to the existence of a special group of nervous disorders following injury, which had to be distinguished from phenomena connected with well-recognised organic affections of the nervous and vascular systems as well as from hysterical manifestations. They are phenomena which are not of a psychical but of a physical nature, and we will now proceed to discuss them.

Can muscular atrophy depend on an hysterical motor disorder? We do not deny that it can, and a priori we ought to be all the more inclined to admit it, as hysterical amyotrophy was first described by Charcot and one of us; but we must add that this amyotrophy is not the rule in hysterical paralysis; that it is slight in degree when it does occur, and that we regard a pronounced muscular atrophy as always due to some other cause.

Charcot and Vulpian long ago insisted on the fact that amyotrophy following an articular lesion cannot be caused by immobilisation. In his Tuesday lectures of 1888 and 1889, which were therefore delivered long after he had given such a remarkable description of hystero-traumatism, Charcot proved that the hypothesis that immobilisation was the cause of the so-called reflex amyotrophy was absolutely untenable,

when the atrophy had developed very rapidly after a lesion of the joint which had required only a few days' rest.

Vincent's cases, in which the atrophy showed no change or even became more pronounced in spite of active mobilisation, constitute a powerful argument against this point of view. In fact, of all the so-called reflex nervous disorders, amyotrophy seems to be the one about which there is least discussion at the present time. Roussy and Boisseau [1] appear to agree with us in this respect. They have recently published the case of a patient in whom amyotrophy of the thigh did not show any appreciable change after a very active treatment of four months' duration, when the circumference of the affected thigh was 6 cm. smaller than that of the opposite side, the difference at the beginning of the treatment having been 7 cm.

If it be granted that the amyotrophy in these cases may be of a reflex character, why should not the same mechanism be accepted in the case of exaggeration of the reflexes? The intervention of a disturbance of the central nervous system appears to be still more certain in the latter than in the former.

It may even be said that exaggeration of the tendon reflexes, observed either in the waking state or in chloroform narcosis, is the most important argument that can be brought forward in support of the reflex pathogeny.

The hyperidrosis which is sometimes observed in these cases cannot be attributed to immobilisation either, and it appears to be the consequence of a stimulation of the nerve centres. André Thomas [2] has arrived at practically the same conclusion in his study of hyperidrosis due to peripheral stimulation. "Whatever may be the mechanism," he writes, "of the perspiration in the three previous cases, its starting-point

[1] Roussy et Boisseau, Sur le pronostic et le traitement des troubles nerveux dits réflexes. *Soc. de Neurol.*, 7 Juin 1917.

[2] André Thomas, Hyperidrose par irritation périphérique. *Soc. de Neurol.*, 9 Nov. 1916, and *Rev. Neurol.*, Nov.–Dec. 1916, p. 544.

is a peripheral irritation, and it implies the involvement of the nerve centres, but probably in a more complicated manner than that of ordinary reflexes." The appearance later of hyperidrosis in the corresponding limb of the opposite side, a phenomenon which we have sometimes seen very clearly displayed, appears to us to constitute a valuable argument in favour of the reflex pathogeny.

It seems to us to be quite as difficult to attribute the pronounced hypotonus seen in some of the cases which we have reported to immobilisation due to hysterical paralysis. It is strange that this argument should be advanced when we remember that about a year ago some neurologists regarded hypotonus as indicating not merely a nervous lesion but an extremely profound lesion of the nerves. Hypotonus, in fact, was considered as one of the signs which formed part of the syndrome of a complete interruption.

We will now discuss the circulatory disturbances which are manifested by cyanosis, hypothermia, and a considerable reduction in the amplitude of the oscillations as measured by Pachon's oscillometer. Is immobilisation alone the cause of this? It would in that case be difficult to explain the fact that hypothermia sometimes alternates with hyperthermia, and that the oscillations after having been at a given moment less extensive on the affected side than on the sound side, may on the contrary become greater on the affected side as the result of the application of heat to the corresponding limbs on both sides simultaneously. It would be still more difficult to explain the fact that in some cases the hypothermia in one region coincides with hyperthermia in another, and that walking can increase hypothermia, as has been shown by Souques. These phenomena fit in well with the idea of a disturbance of the vaso-motor control.

However, whatever the mechanism of these disorders may be, when once they have developed, they are sometimes extremely persistent. Many patients declare that on waking up they feel no difference

between the two sides, but that as soon as they are out of bed, especially if the atmospheric temperature is low, the chilling of the limb appears, and they need hours to get warm again, even in front of the fire. If one of these patients is examined in the morning when his limbs are covered and warm, the symptoms appear to be absent, but when they are uncovered the thermo-asymmetry soon appears. If he is put astride on a stool, with his legs hanging down, and both under the same conditions, the cyanosis appears on the affected side. The obstinacy of these symptoms has been confirmed by Vincent's observations. Like amyotrophy they may resist mobilisation.

It is true that Roussy and Lhermitte [1] in their work on "The Psycho-neuroses of War" made the following statement: "Vaso-motor symptoms, fall of the temperature, cyanosis of the paralysed extremities, hyperidrosis, œdema, etc., are the result of functional inactivity, as is proved by their rapid disappearance when an hysterical paralysis or contracture is cured by psychotherapy."

But in a more recent communication Roussy and Boisseau themselves admit the obstinacy of these disorders, since they express themselves as follows: "As regards disorders of the thermal and vaso-motor control, it has been proved that they may persist for a long time after the recovery of motor power, and improvement, when it does occur, is extremely slow."

When these vaso-motor disorders are pronounced, they give rise to a series of disturbances of a physical character. There may result a diminution or disappearance of the plantar cutaneous reflex, an impairment of the tendon reflexes, especially of the ankle jerk, to which Clovis Vincent [2] has drawn attention, mechanical hyperexcitability of the muscles, and a modification

[1] Roussy et Lhermitte, Psychonévroses de guerre (Collection Horizon), p. 20.

[2] Clovis Vincent, Variations du réflexe achilléen chez certains hommes atteints de troubles physiopathiques des membres inférieurs. *Soc. de Neurol.*, 5 Avril 1917.

of the contraction produced by percussion, which becomes extremely sluggish, and a premature fusion of the faradic contractions. The sluggishness of the muscular contraction produced by percussion, as is shown by the tracings taken in collaboration with Hallion, may be as great as is occasionally seen in the reaction of degeneration.

But the application of heat to the chilled limb is sufficient to cause an immediate reappearance of the plantar cutaneous reflex, an increase in the intensity of the tendon reflexes, which become equal on both sides, a disappearance of the sluggishness of the muscular contraction, and premature faradic tetanus. These circulatory disorders, which we attribute to a reflex vascular spasm, resemble those which are sometimes produced by arterial obliteration.

Oscillometric examination appears to give identical results in both cases. But, as we have shown in the chapter on "Diagnosis," the warm-bath test serves to distinguish the two conditions. Whatever interpretation may be given them, the circulatory disorders which we have studied have a clinical physiognomy of their own, and are entitled to a special place in pathology.

Our opponents will perhaps say: "We readily admit that the symptoms which have just been discussed are quite different from those of pithiatism, and we will even consent to their being considered as of a reflex character or at least of a physiopathic nature, but that does not prove that the motor disorders which accompany them, viz. paralysis and contracture, are of the same character, and we maintain that these phenomena are of a pithiatic nature."

We will now examine these motor disorders and discuss this new aspect of the question. Though we do not dispute the very frequent association of hysterical paralysis or contracture with vaso-motor disorders, as we have ourselves drawn attention to the importance and frequency of this association, we consider that when hypothermia is considerable it causes a diminution of

motor power which is to some extent of a physiopathic nature.

On this point some explanations appear to be necessary. Some medical men in whose minds the word paralysis doubtless suggests the idea of complete or very extensive loss of power, such as hemiplegia, monoplegia or paraplegia, have been surprised by the expression "reflex paralysis" which we have used, and have regarded it as a return to a doctrine which had been given up a long time ago.

The term paralysis, we may say, merely signifies a more or less marked diminution of motor power, without specifying its degree.

Besides, we have expressly stated that there is no such thing as a reflex monoplegia or paraplegia, and that reflex motor disorders are generally limited, partial and incomplete. The fact is that it is impossible to determine in a given case how much is due to the physical factor in the impairment of motor power, but it may be affirmed that this factor enters into play, at least to some extent.

A hypothermia which gives a very decided sensation of cold when the limb is touched and in which the thermometer shows, for instance, a drop of 8° C. as compared with the sound limb, and affects the deep parts as indicated by the slowness of the muscular contraction on mechanical stimulation similar to that observed in the reaction of degeneration, such hypothermia, we say, entails a diminution of the muscular power, or a paresis which is manifested by difficulty in making a prolonged physical effort. A patient who is capable, for instance, of walking quite well for half an hour, but is unable to walk for several hours as he did before, is no longer normal from the motor point of view, and this diminution of his muscular power should be taken into serious consideration, especially in the case of a soldier.

The relation of the phenomena to one another is according to our view quite different from that proposed by our opponents. It is not the hysteria which

comes first, but the disorders of a physical character. The immobilisation due to the traumatism, and the disturbance of the nervous system which is the consequence of this traumatism, give rise to the circulatory disorders and the hypothermia which we have described. These disorders in their turn produce a more or less marked impairment of motor power, which is possibly connected with a sort of torpor of the cells of the anterior cornua of the cord, according to Charcot's hypothesis, and there is no need to attribute all these phenomena to mental disturbance.

If the patient is energetic he will make a great effort to struggle against the disorders which have developed, he will endeavour to put his torpid muscles into action in spite of the discomfort and fatigue which he feels, and indirectly his will can exercise a certain action on his physiopathic symptoms. In the contrary case he will abstain from all effort, and if then suggestion intervenes, pithiatic disorders will be grafted upon the preceding and may even assume a preponderating rôle.

Sometimes all these patients are accused of aboulia; this reproach may be justified in some cases, but in others it is not, for these patients require a strong will or an actual hyperboulia to react with success.

We think that we have definitely proved that the so-called functional contractures are not all pithiatic. The value of our argument, which consists in observations made during chloroform narcosis, does not appear to have been sufficiently appreciated. When a contracture persists, as we have seen, in an advanced stage of narcosis, after obvious loss of consciousness, or when in flexion contracture of the leg on the thigh, it is possible to produce spasmodic movements of the affected limb by traction, although the cutaneous and corneal sensibility are still abolished, it is impossible to admit that we are dealing with a disorder due to suggestion.

We will also remark in this connection that we have never maintained, as some appear to believe, that every flexion contracture of the leg on the thigh is of a

reflex nature, for we are well aware that this form may be caused by hysteria.

In the case of the patients whose photographs we have reproduced, the diagnosis of reflex contracture was made because the spasmodic phenomena persisted until a very advanced stage of narcosis.

It should be noted that in these flexion contractures of the leg the symptoms generally followed wounds of the posterior surface of the thigh; it might, therefore, be urged that the spasm is not of a reflex character, but is due to a direct stimulation of the nerves or muscles; it might be compared with the facial hemispasm, which, as one of us has shown, is due to direct stimulation of the facial. But even admitting that this is so, one would be dealing all the same with a physiopathic contracture, and not an hysterical one, and that is, we repeat, the essential point.

We should like to make it clear that our observations introduce a new idea into neurology. Chloroform narcosis had not been employed before except to distinguish contractures from fibro-tendinous retractions, but it also serves, as we see, to distinguish the various types of contractures.

We should like to say a few words about acro-contracture or accoucheur's hand. We have been credited with the view that all contractures of this kind were due to a disorder of a reflex nature. We have never held such an opinion, but on the contrary have maintained that everything that can be reproduced by the will is within the scope of hysteria. Now, it is quite obvious that the attitude assumed by the accoucheur's hand can be easily reproduced. It is only the deformities which cannot be produced voluntarily that must be excluded from the clinical picture of hysteria; they can only be due to an organic or physiopathic disorder. "But," they may retort, "what is the explanation of the therapeutical results which have been obtained?" They will cause no surprise to those who have carefully read the preceding chapters. We have never regarded reflex disorders

as incurable, and have even definitely expressed the opposite opinion. We have said, it is true, that physiopathic disorders are generally obstinate, but chiefly in comparison with hysterical disorders, which sometimes disappear almost instantaneously under the influence of counter-suggestion. The occasionally very rapid disappearance of the motor symptoms associated with physiopathic phenomena is readily explained by the frequency of the association of hysteria with every other affection.

It appears, however, that this very simple notion has not been understood by some medical men, or at least that they have not grasped its complete significance. As regards hystero-reflex associations in particular, they are extremely common, and we have given several examples of them in the chapter on "Diagnosis."

We were so convinced of this truth in writing our book that we expressly recommended that these patients should undergo psychotherapeutic treatment, even in cases in which the reflex symptoms were most characteristic.

To regard as permanent, or even as necessarily obstinate, all the motor disorders associated with some physiopathic phenomenon would be in practice as grave an error as to attribute them all indiscriminately to hysteria.

It would indeed be a gross and wilful exaggeration of our ideas, which do not in any way justify such conclusions. We can hardly admit even that the so-called reflex disorders can last beyond a certain period, even when they are very marked. Cases of amyotrophy of the quadriceps following arthritis of the knee have been known to last for years, and to show changes for the better or the worse, but the latter is due to a recrudescence of the joint affection. When the causal lesion is definitely cured, the motor disorders and other physiopathic symptoms ought to be cured also.

If they persist it is doubtless because they are fixed

by the psychopathic element, which was first associated with them and then took their place, the motor disorders of paralysis and contracture having become purely hysterical, and the residue of the other symptoms, such as atrophy, hypotonus and circulatory disorders, being kept up by immobilisation.

We have now to determine the possible duration of this stage in which the motor disorder is reflex. As a matter of fact we have no certain knowledge on this point. The duration sometimes appears to be fairly long; in several patients who had been examined during chloroform narcosis more than a year after the onset of the symptoms, their contracture persisted until an advanced stage of the narcosis, which indicated its physiopathic nature. But there is every reason to suppose that such cases are relatively rare, and that the substitution of pithiatic phenomena for reflex symptoms may be rapid. A patient therefore who may resist psychotherapy at one period may be amenable to it later.

This is one of the reasons for which the following clause was contained in the conclusions drawn up by one of us, and adopted by the Neurological Society on April 7, 1916, to terminate the discussion on so-called functional nervous disorders.

"Before any decision is taken by a medical board it is important that the wounded suffering from this variety of motor disorders should undergo a prolonged examination in a neurological centre, and that all the appropriate psychotherapeutic and physiotherapeutic methods should be employed simultaneously. . . . Leave for convalescence may be granted, but on condition that the patients on the expiration of their leave are sent back to the same neurological centre for examination and fresh treatment."

We understood that some medical men are inclined to regard as incurable patients suffering from traumatic motor symptoms when they present some physiopathic phenomena such as hypothermia, more or less marked hypotonus or slight amyotrophy, and that patients

of this kind are too often recommended for discharge from the service. We regret that this is so, but nothing of what we have written justifies such a procedure. This constitutes, however, a danger against which we have warned medical men who were not sufficiently alive to it.[1]

At the beginning of this postscript we provisionally admitted that the great majority of patients with physiopathic disorders were capable of recovering after three or four months' treatment, and we have just shown that if this was correct, as some of our opponents mentioned, it would not be in opposition to the ideas which we have set forth. It remains clearly established that there is a group of nervous disorders comprising contractures and paretic states, occupying, as it were, an intermediate position between organic affections properly speaking and hysterical phenomena.

But are we justified in asserting that patients with nervous disorders of this kind, even under the most favourable conditions, can be cured almost infallibly in a few months? Such optimism would not be justified, for careful observations undoubtedly prove that the duration of physiopathic phenomena may be much longer. Clovis Vincent's observations which we have quoted are most conclusive in this respect.

In a recent communication our colleague brought forward some equally conclusive cases which prove the obstinacy of physiopathic symptoms in some cases. The military distinction which he gained, his remarkable success in the treatment of inveterate hysterical symptoms, his energy and perseverance, all confer considerable value on his observations. What makes them still more conclusive is that Clovis Vincent was convinced at first, as he recently stated,[2] that

[1] J. Babinski et J. Froment, Troubles physiopathiques d'ordre réflexe—Association avec l'Hystérie—Evolution—Mesures Médico-militaires. *Presse Médicale*, 9 Juillet 1917.

[2] Clovis Vincent, Sur le traitement et le pronostic des phénomènes physiopathiques. A propos de la communication de MM. Roussy, Boisseau et d'Oelsnitz. *Soc. de Neurol.*, 7 Juin 1917.

the so-called reflex disorders would not resist the treatment which he had adopted at Tours, and in which he had simultaneously employed rapid psychotherapy, energetic galvanism, intensive re-education, and methodical exercises and training.

For several months he made the patients suffering from disorders of this kind carry out these exercises under his personal supervision, and his long experience led him to conclude that unlike pithiatic phenomena reflex nervous disorders are very obstinate, and may resist the most energetic treatment and a training greatly exceeding in duration the time previously fixed.

Moreover, even those of our opponents who are most convinced of the efficacy of combined psychical and physical measures in the treatment of so-called physiopathic disorders have been led to declare recently that a cure, *i.e.*, a return to a completely normal condition, is far from constant, and that the patients in question, even if they can be cured, often remain unfit for military service.[1]

This does not mean that complete recovery from the most pronounced physiopathic disorders of a reflex character is impossible in the more or less distant future. We have seen a slow and progressive disappearance of contractures which during chloroform anæsthesia had presented the following characters: persistence of the contracture until an advanced stage of narcosis, and spasmodic movements of the affected limb caused by any attempt to correct the vicious attitude.

As regards determining the proportion of cases in which a complete cure will require a long time as compared with those in which recovery is fairly rapid, and fixing the maximum period necessary to obtain complete return to the normal condition, these, it must be confessed, are questions which our present experience does not yet allow us to answer.

We have one more remark which we should like to

[1] Roussy et Boisseau, *Soc. de Neurol.*, 5 Juillet 1917.

make. We have been confronted with the objection that the so-called physiopathic disorders are complex, and that care must be taken to avoid mixing them all up together. Possibly distinctions will have to be made later in this group, but the separation made by us far from being a confusion constitutes a dissociation, and we should be inclined to apply to our opponents the criticism which they have addressed to us. We have often had occasion to see patients with physiopathic disorders of a reflex character, whom other observers without any solid argument to support their diagnosis had considered as suffering from neuritis, arteritis or merely fibro-tendinous retractions. Sometimes the diagnosis of neuritis had been solely based on the slowness of the contraction caused by percussion of the muscles, although the characteristic signs of the reaction of degeneration were absent, and there were no motor or sensory disorders with a peripheral distribution; in some cases the mere presence of cyanosis or very marked hypothermia had led to the diagnosis of an arterial lesion without any measurement of the blood-pressure or any attempt to exclude the hypothesis of reflex vaso-motor disorders by the hot-bath experiment; in other cases a reflex contracture was not recognised because the vicious attitude was attributed to fibro-tendinous retractions, which, as was shown by examination under chloroform, were either non-existent or were associated with a contracture which was mainly responsible for the deformity.

Such errors and confusions led to the elimination from the group of physiopathic disorders of just those very ones which in our opinion were the most characteristic, and which offer most resistance to treatment. It is to cases of this kind that the term physiopathic is most applicable: a psychopathic or pithiatic state cannot explain them and although there is a justification for the statement that the condition is due to physical changes, none of the pathognomonic physical signs of lesions of the nervous or vascular systems are found; if such lesions exist they are too limited

to account for the disorders present. We are well aware of the existence of those complex cases in which so-called reflex disorders are associated with a disturbance of the activity of the wounded limb resulting from a lesion of a nerve trunk, or changes in the muscles, tendons and bones, accompanied by cicatricial adhesions which are so common after wounds in war. The interpretation of such cases, as we have already shown, is sometimes a very difficult matter.

If, on the one hand, the group of so-called reflex disorders has the most marked and consequently the most obstinate symptoms eliminated from it, and if, on the other hand, motor disorders are introduced, accompanied by only very slight physiopathic phenomena which in our opinion are mostly of a pithiatic nature, a necessarily false conception is obtained of those physiopathic phenomena known as reflex.

This is perhaps the motive which has led some neurologists to refuse to accept the ideas which we have developed, although they recognise the correctness of the various new facts which our researches have enabled us to establish.

July 1917.

APPENDIX

REFLEX NERVOUS DISORDERS

Considerations of space prevent us from giving a full account of the numerous cases of contracture and paralysis on which our description of reflex nervous disorders is based. We will confine ourselves to giving a summary of some of our observations dealing in the first place with the state of the tendon reflexes, and secondly with the state of the peripheral circulation and the results of our sphygmometric and oscillometric examinations.

1. Observations made during Chloroform Narcosis

A. *Exaggeration of the tendon reflexes of the traumatised limb in reflex contractures*

It may be noted, first of all, that normally the stage of exaggeration of the tendon reflexes in chloroform anæsthesia appeared to us to be inconstant; in any case, it is ill marked. The order in which the reflexes disappear and reappear varies; sometimes the disappearance of the plantar cutaneous reflex precedes disappearance of the tendon reflexes and sometimes follows it; sometimes also, as we have noted, the evolution of the phenomena is still more complex. Thus in a patient who had been operated on for a tumour of the breast under chloroform, we found on her coming round from the anæsthetic that the reflexes reappeared in the following order: ankle jerk, plantar cutaneous reflex, knee jerk, elbow jerk; the disappearance of the reflexes had been too rapid to allow us to follow the

various stages. There are, therefore, very great individual differences between one case and another, but normally the phenomena of the tendon reflexes including their disappearance, return, diminution and exaggeration, always appeared to us to be symmetrical (122). The following are our cases relating to reflex contractures (125)—

Case I.—L. . . . Shrapnel wound which had involved the upper and external part of the right thigh. Contracture of pelvi-trochanteric muscles. *The right knee jerk is a little more marked than the left; the difference is very slight,* but constant, having been found again at several days' interval. After a few stimuli the right knee jerk becomes even a little polykinetic. The ankle jerks are normal and equal. There is no ankle or patellar clonus.

Chloroform anæsthesia, September 20, 1915.—From the commencement of the anæsthesia and even before the stage of motor excitement and struggling exaggeration of the two knee jerks and a faint patellar clonus on both sides have been found; but while the excitement rapidly diminishes on the left, it progressively increases on the right. During complete muscular relaxation when all the other tendon reflexes (knee and ankle jerks on the left, and wrist and elbow jerks on both sides) are lost, a very definite patellar clonus can be obtained on the right. The reflex obtained by percussion of the patellar tendon is strong and polykinetic. The right ankle jerk is preserved but not exaggerated. On coming round from the anæsthetic, at the time when the reflexes are returning, a slight patellar clonus on the left is found; the right clonus is as marked as before. At no stage did we meet with ankle clonus. *Right patellar clonus continued for an hour after coming round.* The reflexes then returned to the state in which they had been before the anæsthesia.

Case II.—Corporal B. . . . Wounded April 22, 1915, by a bullet which penetrated the scrotum and came out through the middle of the right buttock. The urethra was injured, and the wound required fully four months to cicatrise. Contracture of pelvi-trochanteric muscles. *The right knee jerk seems a little stronger and of wider range than the left, but this difference is very slight and it is difficult to declare that*

the patient does not increase the amplitude of the movements caused by percussion of the tendon.

Chloroform anæsthesia, October 10, 1915.—No stage of definite exaggeration of the tendon reflexes found when the patient went under the anæsthetic. The tendon reflexes became diminished and then disappeared very early. On coming round the plantar cutaneous reflexes appeared long before the knee jerks. *The right knee jerk appeared distinctly and a fairly long time before the left. It was already brisk when hardly any muscular contraction was detected in the left.*

Case III.—Soldier V. . . . as the result of a fall, complained of pain in the right hip and presents claudication. Contracture of pelvi-trochanteric muscles. Right knee jerk a little the stronger, perhaps slightly polykinetic, *but the difference between the two knee jerks is very slight.*

Chloroform anæsthesia, September 5, 1915.—As soon as the patient went under the anæsthetic the knee jerks became more marked without there being any general exaggeration of the tendon reflexes. *After termination of the anæsthesia and during the stage of reappearance of the reflexes the right knee jerk became polykinetic, and patellar clonus on the same side was obtained of short duration but definite and typical.* The difference between the two knee jerks was much more marked than in the waking state.

Case IV.—Soldier L. . . . was hit by a bullet which penetrated the inner surface of the root of the right thigh and came out at the level of the great trochanter, after causing a fracture of the upper extremity of the femur. Contracture of the pelvi-trochanteric muscles. Both knee jerks are somewhat brisk, but *the right knee jerk on careful examination seems a little stronger than the other.* Sudden depression of the patella causes a faint clonus on the right, but it is difficult to be certain about its nature and to assert that it is a true clonus.

Chloroform anæsthesia, September 10, 1915.—During the stage of motor excitement exaggeration of the tendon reflexes was not observed at any moment, and the faint clonus noted in the right patella disappeared. The radial and olecranon reflexes completely disappear. It is the same

with the left knee jerk, but its disappearance is less definite. Reduction in the degree of anæsthesia is sufficient to make it reappear. *It is not, however, possible to make the right knee jerk disappear completely.* During the stage of recovery from the anæsthetic the difference between the two knee jerks is well marked; *the right knee jerk is much the strongest*, the right leg describes a more extensive movement, and on falling back strikes the operation table with greater suddenness and violence. Even to an observer who does not see it but merely hears the noise produced, the difference is considerable. Sudden depression of the patella causes a slight *clonus on the right*, which can also be produced by suddenly raising the thigh and letting the leg fall by its own weight. *The asymmetry in the reflexes which was doubtful in the waking state is thus made quite obvious.*

Case V.—Lieutenant M. . . . aged 33. Bruise from shell fragment, October 6, 1914. Contracture of the right pelvi-trochanteric muscles. *Both knee jerks are brisk, but the right is slightly more so than the other.* The muscular contraction is stronger and lasts longer, with a slight tendency to polykinesis. There is no patellar clonus nor ankle clonus, and the ankle jerks are normal.

Chloroform anæsthesia, August 15, 1915.—Although *all the other reflexes* (tendo Achillis, radial, olecranon, plantar cutaneous and corneal), *including the left knee jerk, are abolished, a very marked right patellar clonus is observed.* To produce this clonus it is not necessary to employ the ordinary method; it is enough to raise the limb and let it fall by its own weight. The right knee jerk is very brisk and very definitely polykinetic. On recovery from the anæsthetic, the patellar clonus becomes bilateral, but remains very unequal. It can scarcely be detected on the left, whereas it is extremely definite and brisk on the right. After recovery from the anæsthetic the patellar clonus persists with the same characters for about an hour and a half, and the ankle clonus for about three hours.

Case VI.—Soldier D. A. . . . aged 26, was hit by a bullet in the right calf in September 22, 1914. There was no fracture as was shown by the X-rays, but cicatrisation was slow and took no less than three months. *The right*

knee jerk is a little stronger and more sudden than the left, but the difference is very slight, and there is some doubt as to whether this exaggeration really does exist. *The difference between the two ankle jerks is even more doubtful:* all that can be said is that if there really is one, the balance is in favour of the right side.

Chloroform anæsthesia, October 1915.—As soon as the patient lost consciousness, the following phenomena were noted: both knee jerks and the left ankle jerk disappeared even before the end of the stage of motor excitement and restlessness. They rapidly became weaker before disappearing, and did not show any stage of exaggeration any more than the other tendon reflexes The anæsthesia was then stopped: *the right ankle jerk which had never disappeared was still definite and even more marked than in the normal condition and was polykinetic.* During the stage of recovery from the chloroform the right ankle jerk remains brisk and polykinetic without, however, any ankle clonus. *The difference between the two ankle jerks is very distinct and admits of no doubt.* The period of distinct asymmetry of the knee and ankle jerks lasted about ten minutes after cessation of the anæsthesia, a little longer in the case of the knee jerks than in that of the ankle jerks.

Case VII.—B. . . . Typhoid fever, October 20, 1914; phlebitis, abscess in right buttock. Contracture of pelvi-trochanteric muscles. Sent to the out-patient department at La Pitié for a second report to be made on him. We found the following notes in the patient's case sheet—

"*Examination on September* 22, 1915.—The patient shows a slight laxity of the patellar tendon, but as much on the left as on the right. The right side is more cyanosed, which is due to the inactivity of the limb. There is no œdema. *Tendon and cutaneous reflexes normal.*

"*The loss of power is entirely functional. The patient will begin to walk as soon as he wants to.*"

In the examination at La Pitié we found that the knee jerks were brisk and polykinetic; *the right knee jerk seemed to be a little more marked than the left.*

Chloroform anæsthesia, October 25, 1915.—The knee jerks, ankle jerks, plantar cutaneous and cremasteric re-

flexes, disappeared almost instantaneously. During the first stage of anæsthesia no increase in the asymmetry of the reflexes was noted.

At the commencement of the period of recovery *an early appearance of a very definite right knee jerk was noted, while the left knee jerk was still lost.* At a later stage of recovery, the right knee jerk suddenly showed a very definite exaggeration, and on the right, but *on the right only there was patellar clonus.* Percussion of the left patellar tendon caused a contraction of the right adductors, and at the time when the exaggeration of the reflexes was most marked an actual tonic and clonic spasm in these muscles. Percussion of the right patellar tendon did not cause any contraction of the right or left adductors. Ankle clonus was never observed.

Case VIII.—Soldier P. . . . Typhoid fever, November 25, 1914. Abscess at the root of the left thigh. Contracture of the left pelvi-trochanteric muscles. *The knee jerks are both brisk, but the left is a little brisker than the right.* There is no patellar clonus. Percussion of the left tendo Achillis causes a slow and sustained contracture of the gastrocnemius.

Choloroform anæsthesia, October 25, 1915.—An obvious increase in the asymmetry of the knee jerks was noted from the commencement of the anæsthesia about four minutes after the patient had begun to take the chloroform and while he was still struggling and reacting to a prick; the left knee jerk was much more brisk than the right, but there was never any patellar clonus. *An exaggeration of the ankle jerk with ankle clonus* was also observed on the left side. Six minutes after the commencement of anæsthesia the right knee jerk was lost, the right ankle jerk extremely weakened, and the same applied to the left knee jerk. *Ankle clonus in the left foot was present throughout the recovery stage, and was also quite as marked three-quarters of an hour after complete recovery.*

Case IX.—Soldier P. . . . Wounded September 8, 1914, by a fragment of shell, which entered the upper part of the right buttock and lodged in the sacro-coccygeal region, from which it was removed. *The knee and ankle jerks*

seemed to be a little brisker on the right, but the exaggeration was doubtful.

Chloroform anæsthesia, October 15, 1915.—*Throughout the anæsthesia a decided unilateral exaggeration of the right knee jerk with unilateral patellar clonus was observed.* The left knee jerk never showed any obvious exaggeration, but grew progressively weaker. (The anæsthetic was not continued until it disappeared.) The ankle jerks grew weaker and disappeared; ankle clonus was never observed.

Case X.—Soldier F. . . . Wound of the left arm August 22, 1915, followed by cellulitis. Contracture of upper limb in position of extension with accoucheur's hand No exaggeration of the tendon reflexes in the affected hand.

Chloroform anæsthesia.—The contracture did not persist till an advanced stage of the narcosis, but an *exaggeration of the left ulnar pronator reflex with tendency to clonus was observed.*

B. *Persistence of the contracture to an advanced stage of narcosis*

Case XI.—L. . . . Bullet wound of left ankle, September 1, 1914. Contracture of foot and last four toes in position of extension with flaccid paresis of the great toe (*v.* Pl. VIII, p. 578). The left knee jerk was a little brisker than the right; the left ankle jerk appeared weaker, but the contracture of the foot made this difficult to decide.

Chloroform anæsthesia, October 22, 1915.—No definite asymmetry of the tendon reflexes except that the left ankle jerk appeared somewhat weaker than the right. The contracture disappeared completely during the stage of muscular relaxation, but reappeared soon after the return of the tendon reflexes and continued henceforward without any change. *The reappearance of the contracture preceded the return of consciousness by twenty to twenty-five minutes.*

Case XII.—B. . . . Wounded on November 11, 1914, in the right thigh (middle third), flexion contracture of leg (*v.* Pl. I, p 502). The right knee jerk was normal, and the right ankle jerk a little weaker than the left.

Chloroform anæsthesia, November 2, 1915.—The contracture disappeared completely during the stage of muscu-

lar relaxation, but reappeared at the same time as the right knee and ankle jerks as soon as the administration of chloroform was stopped. *The reappearance of the contracture definitely preceded the return of sensibility as well as the return of the cutaneous reflexes and conjunctival reflex, and was already complete twenty minutes, at least, before consciousness returned.* Slight asymmetry of the tendon reflexes during the stage of recovery.

Case XIII.—Soldier J. Pl. . . . Wound of the posterior surface of the left thigh, September 6, 1914. Contracture of the leg in a position of hyperextension on the thigh, and contracture of the foot in a position of equino-varus (*v.* Fig. 12, p. 556).

Chloroform anæsthesia, September 12, 1916.—The deformity of the leg and foot diminished during the stage of muscular resolution, but did not disappear completely. Very pronounced fibrous retractions were therefore associated with the contracture.

The contracture reappeared very definitely before the return of consciousness. The left knee jerk reappeared before the right, and left patellar clonus was then found.. At a more advanced stage of recovery it was possible to produce clonus on the right, but the phenomenon was much less marked than on the left.

C. *Spasm produced during narcosis*

Case XIV.—Captain B. . . . Wounded in the posterior part of the thigh. Paralysis of the sciatic with flexion contracture of the leg on the thigh.

Chloroform anæsthesia, October 15, 1915.—The contracture disappeared completely in the period of muscular relaxation, *i. e.*, twenty-five minutes after the commencement of the anæsthesia, while consciousness disappeared in eight minutes. Its disappearance and that of the plantar cutaneous reflex were almost simultaneous. During complete anæsthesia any effort to correct the vicious attitude caused a movement of flexion of the leg resembling that produced by cutaneous stimulation in cutaneo-reflex contractures, but here the cutaneous stimulation was ineffec-

tive. The spasm which was not present in the waking state did not return in the stage of muscular relaxation; it disappeared and reappeared at the same time as the plantar cutaneous reflex [1] and the contracture; it could be provoked about ten minutes before the return of the first manifestations of consciousness.

Sphygmometric and Oscillometric Observations

As will be seen in the annexed tables which contain a summary of some of our observations, our researches (161) deal with the state of the peripheral circulation in various classes of cases,[2] including reflex nervous disorders, lesions of the nerve trunks, lesions of the large vessels, and various associations of nerve disorders. This comparative study has enabled us to estimate better the sphygmometric and oscillometric character of the circulatory disturbances in reflex paralysis and contractures.

[1] In a woman who showed a cutaneo-reflex contracture with flexion paraplegia, whom we had the opportunity of examining during chloroform narcosis, the disappearance as well as the return of the reflexes of defence and plantar cutaneous reflexes were equally simultaneous.

[2] In their interesting study on "the real symptomatology of complete section of peripheral mixed nerves," H. Claude and Stephen Chauvet (195) studied the vaso-motor reactions in complete section of the peripheral mixed nerves. They referred to the researches of Lapinski, who had observed the three following stages after experimental section: 1. A short stage of arterial constriction. 2. A stage of marked vaso-dilatation lasting 26 to 60 hours. 3. A stage in which the intensity of the vaso-dilatation diminished although it still remained quite distinct. In the clinical cases of recent nerve sections (less than a month) which they had the opportunity of seeing they noted vaso-dilatation; we should state that they based the diagnosis of vaso-dilatation simply on the redness of the skin. It should be added that in order to make this redness more distinct in certain cases they recognised the necessity of examining the patient after he had been in a warm room, and let his hand hang down for a few moments when the upper limb was affected. Lastly, they noted that this vaso-dilatation was not of indefinite duration, and that even in the absence of a union of the nerves the normal vascular tonicity was likely to return.

TABLE I

Name.	Reflex Disorders of Upper Limb without Lesion of Nerve Trunks.		Systolic Pressure. Riva-Rocci-Vaquez.	Oscillations. Brachial	Oscillations. Radial.	Gaertner on Index.	Date of Examination.
I. R . . .	Wound of left shoulder, August 1914; atrophy, cyanosis, hypothermia, reflex paresis associated with hysteria.	Sound side	14·5	18°	3°	11	Nov. 1915
		Affected side	14·5	15°	1°	3	
II. M . . .	Through and through wound of left forearm without fracture, December 1914; atrophy, cyanosis, tendinous retractions, paralysis of muscles of forearm and hand, very pronounced hypothermia, exaggeration and slowness of mechanical contractility.	Sound side	14·5	24°	7°	9	Feb. 24 1916
		Affected side	13·5	20°	1°	0	
		Sound side	12·5	20°	6°		Mar. 8 1916
		Affected side	11·5	14°	0°·5		
III. B . . .	Through and through wound of left forearm, August 1915; slight cone-shaped contracture of hand, variable disturbances of temperature.	Sound side	10·5	14°	8°	9	Feb. 1916
		Affected side	10·5	14°	8°	8	
IV. D . . .	Through and through wound and fracture of left carpus, November 1914; very pronounced cone-shaped contracture of hand, slight hypothermia, exaggeration of mechanical contractility.	Sound side	13	18°	6°	15	Dec. 1915
		Affected side	13	16°	4°	11·5	

V. P . . .	Wound of antero-inferior part of right axilla, January 1915; slight contracture of hand, slight hypothermia.	Sound side	14°	26°	14°	10·5	April 1916
		Affected side	14·5	26°	7°	9	
VI. M . . .	Wound of left arm, September 1914; bicipital retraction, paresis, slight hypothermia.	Sound side	14·5	20°	6°	14	Feb. 1916
		Affected side	14	17°	4°	11	
VII. B . . .	Wound of right forearm, January 1915; retraction of flexor tendons, hypothermia, exaggeration of mechanical contractility, slight paresis of hand.	Sound side	13·2	20°	6°	10	Jan. 1916
		Affected side	13	20°	2°	4	
VIII. A . . .	Through and through wound of left wrist, April 1915; paralysis of muscles of forearm and hand, very pronounced hypothermia.	Sound side	11·5	20°	5°	7·5	Mar. 1916
		Affected side	11·5	20°	3°	4	
IX. M . . .	Dislocation and fracture of left elbow, April 1915; total paralysis of muscles of forearm and hand, diffuse redness of limb, hypothermia and mechanical contractility very pronounced on certain days, absent on others.	Sound side	14	24°	4°	12·5	Mar. 2 1916
		Affected side	14	24°	2°	10	
		Sound side			5°		Mar. 6 1916
		Affected side			6°·5		

TABLE I—(*continued*)

Name.	Reflex Disorders of Upper Limb without Lesion of Nerve Trunks.		Systolic Pressure. Riva-Rocci-Vaquez.	Oscillations.		Gaertner ou Index.	Date of Examination.
				Brachial.	Radial.		
X. M . . .	Wound of left arm, June 1915; paralysis of hand with pronounced hypothermia.	Sound side	14	16°	5°	5	Mar. 1916
		Affected side	13	14°	3°	5	
XI. F . . .	Wound of left forearm, August 1914; paralysis of extensors of hand, variable disturbance of temperature.	Sound side	13	14°	2°·5	7	Mar. 1916
		Affected side	12	12°	0°·5	5	
XII. P . . .	Wound of anterior surface of left forearm; paresis of forearm and hand, hypothermia.	Sound side	16	28°	8°	11	Mar. 1916
		Affected side	15·7	24°	5°	7	
XIII. B . . .	Fracture of neck of humerus by bullet, September 1914; paresis of arm, hypothermia.	Sound side	11	22°	4°	9	May 1916
		Affected side	11	14°	2°	8	
XIV. S . . .	Through and through wound of right wrist, September 1914; contracture of hand with flexion, hypothermia.	Sound side	14·5	16°	11°		Dec. 1915
		Affected side	14·5	14°	6°		

Name.	Reflex Disorders of Lower Limb without Lesion of Nerve Trunks.		Systolic Pressure (Riva-Rocci-Vaquez) in dorsalis pedis.	Oscillations. Femoral.	Oscillations. Tibial.	Gaertner Second Toe.	Date of Examination.
XV. M . . .	Traumatism from falling in of trench, November 1914; cyanosis, œdema, and extreme coldness of left lower limb, paralysis, exaggeration of mechanical contracture.	Sound side	12		4°		Sept. 1915
		Affected side	0 ?		1°		
		Sound side	12	26°	8°	3	May 1916
		Affected side	10 ?	14°	1°	0	
XVI. V . . .	Wound of right leg, September 1915; slight paralysis, coldness, exaggeration of mechanical contractility and tendon reflexes.	Sound side	16	14°	11°	8·5	April 1916
		Affected side	16	12°	6°	7	
XVII. D . . .	Wound of left leg. September 1915; paresis of leg and foot, exaggeration of mechanical contractility.	Sound side	12·5	20°	10°	12	Mar. 1916
		Affected side	10 ?	14°	2° 5	6	
XVIII. L . . .	Wound of right calf, November 1914; contracture of right knee, muscular atrophy.	Sound side	15	18°	10°		Mar. 1916
		Affected side	14	16°	3°		
XIX. D . . .	Fracture of right tibia, muscular weakness of leg, very marked hypothermia, exaggeration of mechanical contractility.	Sound side	12		12°		May 1916
		Affected side	?		2°		

TABLE II—(*continued*)

Name.	Reflex Disorders of Lower Limb without Lesion of Nerve Trunks.		Systolic Pressure (Riva-Rocci-Vaquez) in dorsalis pedis.	Oscillations.		Gaertner. Second Toe.	Date of Examination.
				Femoral.	Tibial.		
XX. B . . .	Contusion of right lower limb from shell explosion, December 1916; paresis of limb with hypothermia and exaggeration of mechanical contractility with modification of electrical reactions.	Sound side	14·0	14°	12°	15	June 1916
		Affected side	13·5	11°	6°	9	
XXI. G . . .	Wound of metatarso-phalangeal joint of little finger, August 1914; atrophy, coldness, paresis of right leg and foot, exaggeration of mechanical contractility.	Sound side	12·5		10°		Oct. 1915
		Affected side	12		7°		
		Sound side		20°	14°		Mar. 1916
		Affected side		18°	7°		
XXII. B . . .	Wound of right thigh, November 1914; contracture of knee persisting during light chloroform narcosis, no hypothermia nor exaggeration of mechanical contractility in foot.	Sound side	14		8°		Jan. 1916
		Affected side	14		8°		

XXIII. V . . .	Wound of calf, November 1914; with adherent scar; slight contracture of left knee, no hypothermia nor exaggeration of mechanical contractility except on posterior surface of thigh.	Sound side	14		4°·5		July 1915
		Affected side	14		4°		
XXIV. L . . .	Slight wound of dorsum of left foot, end of October 1914; paralysis of extensors of toes and tibialis anticus, coldness of leg, exaggeration of mechanical contractility of leg and foot muscles.	Sound side	12	24°	7°	11	Mar. 1916
		Affected side	12	20°	4°	8	
XXV. L . . .	Wound of posterior surface of right thigh, contracture of knee with hypothermia and exaggeration of mechanical contractility of leg and foot muscles.	Sound side	14·5	16°	5°		April 1916
		Affected side	14·5	14°	4°		
XXVI. R . . .	Commotion from shell explosion, April 1914; paralysis of right leg and foot with hypothermia and exaggeration of mechanical contractility (hystero-reflex association).	Sound side	12		6°		Feb. 1916
		Affected side	12		2°		

TABLE II—(*continued*)

Name.	Reflex Disorders of Lower Limb without Lesion of Nerve Trunks.		Systolic Pressure (Riva-Rocci-Vaquez) in dorsalis pedis.	Oscillations. Femoral.	Oscillations. Tibial.	Gaertner. Second Toe.	Date of Examination.
XXVII. A . . .	Slight wound of left leg and commotion from shell explosion, September 1914; paralysis of left lower limb with hypothermia and exaggeration of mechanical contractility (hystero-reflex association).	Sound side	16	18°	12°	8°	Dec. 1915
		Affected side	16	14°	8°	6°	
XXVIII. M . . .	Seton in left buttock, September 28, 1914; hypothermia of limb, exaggeration of mechanical contractility, contracture of knee and toes in flexed position.	Sound side	13·5		18°		May 1916
		Affected side	13		14°		
XXIX. G . . .	Trench foot in winter of 1914–15, wound of left leg by 3 shell fragments, left lower limb weak and atrophied, hypothermia especially in winter, cyanosis, exaggeration of knee jerk, and mechanical contractility of muscles.	Sound side	14		9°		Mar. 1916
		Affected side	14		2°		
		Sound side	14		18°	12·5	July 1916
		Affected side	14		12°	11·5	

As may be seen on reference to Tables I and II, the systolic pressure is not modified in the limb affected with reflex nervous disorders, but the amplitude of the oscillations in the radial or in the tibial arteries is weaker, and the arterio-capillary tension is lower than in the sound limb [1] This asymmetry is so obvious and so frequently obscured, at least in cold weather, that one is justified in regarding it as one of the characteristic symptoms of the nervous disorders under consideration. So long as the conditions under which the observations are made remain the same, it may be found in the same patient at each examination (Cases II, XV, XXI and XXIX), but its intensity may vary considerably from one month to another. A rise in the atmospheric temperature or a prolonged stay in a hot room is sufficient to cause a disappearance of the differences previously noted (Case IX). Even in cases where this asymmetry is very marked, a hot bath, as we shall see later (p. 690), diminishes it (Cases II, XI and XV), or causes it to disappear (Cases IV, VIII, XVI and XXVI); sometimes even this asymmetry is reversed (Case IX)

The diminution of the amplitude of the oscillations and of the arterio-capillary tension in the wounded limb, in view of its instability and changeable character, can only be explained by a vascular spasm which occurs whenever the atmospheric temperature is low. This indicates, as we have shown, an actual disturbance of the local vaso-motor control. The wounded limb behaves as if it were specially sensitive to the action of thermal agents, and as if it were unable to maintain normal control over its circulation except under particularly favourable atmospheric conditions.

We will now relate the results of our further researches, which may be classified as follows :—

Table III.—Lesions of nerve trunks without the association of definite reflex nervous disorders. Tables IV and V.—Reflex nervous disorders associated with a lesion of a nerve trunk. Tables VI and VII.—Arterial lesions associated with lesions of nerve trunks and reflex nervous disorders.

[1] All these examinations were carried out with the same Pachon s instrument which had already been used for the study of oscillations in the normal individual. This detail deserves to be noted, as different instruments do not always register oscillations of equal amplitude in the same individual, and the oscillometers on sale at the present time show less ample oscillations than those of the old pattern, such as the one which we employed.

TABLE III

Name.	Lesion of Nerve Trunks without Association of Definite Reflex Nervous Disorders.		Systolic Pressure Riva-Rocci-Vaquez.	Oscillations.		Gaertner.		Date of Examination.
				Brachial.	Radial.	Index.	Ring Finger.	
XXX. P . . .	Wound of left arm, December 1914; section of ulnar nerve severed by operation (complete R.D., anæsthesia), no reflex disorder, no hypothermia, no exaggeration of mechanical contractility, no tendinous retractions.	Sound side	12	14°	12°			Feb. 1916
		Affected side	12	14°	12°			
		Sound side	12	16°	8°	11·5	11	July 1916
		Affected side	12	17°	8°	11	11	
XXXI. A . . .	Cellulitis of the hand with lesion of right median nerve (partial R.D. very marked), no vaso-motor trouble, no mechanical hyperexcitability of muscles.	Sound side	12	10°	7°	13	13	Mar. 1916
		Affected side	12	12°	7°	13	13	
XXXII. V . . .	Section of 5th left cervical root, May 1915; anæsthesia in region of circumflex and musculo-cutaneous, total R.D. of deltoid, biceps, brachialis anticus, and supinator longus; hypothermia limited to upper arm and forearm, no exaggeration of mechanical contractility of muscles of forearm and hand.	Sound side	11·5	18°	4°	11	13·5	June 1916
		Affected side	11	14°	6°	11·5	13	

XXXIII. M . . .	Wound in upper part of left arm with perforation of median nerve, July 10, 1916; large swelling in the course of the nerve, atrophy and partial R.D. of muscles innervated by median, hypæsthesia in region of this nerve, no reflex motor disorders, no hypothermia nor changes in colour of the skin.	Sound side	15·5	20°	8°	15	16	Aug. 1916
		Affected side	15·5	16°	8°	15	15	
XXXIV. L . . .	Wound of right thigh (lower third), September 1915; slight lesion of internal popliteal (pains, hypæsthesia, partial R.D.); hypothermia only in leg, no increase of mechanical excitability.	Sound side	16	22°	12°	10		June 1916
		Affected side	16	18°	8°	12		
XXXV. A . . .	Wound of left leg, June 1915; section of external popliteal (complete R.D.) very moderate hypothermia, no modifications of mechanical contractility, normal reflexes.	Sound side	12	12°	6°	5		Jan. 1916
		Affected side	12	14°	6°	5		

TABLE III—(*continued*)

Name.	Lesion of Nerve Trunks without Association of Definite Reflex Nervous Disorders.		Systolic Pressure Riva-Rocci-Vaquez.	Oscillations.		Gaertner.		Date of Examination.
				Brachial.	Radial.	Index.	Ring Finger.	
XXXVI. F . . .	Wound in popliteal space, March 1916; lesion of external popliteal (hypæsthesia, complete R.D.), no reflex disorders.	Sound side	14	16°	8°	9		June 1916
		Affected side	14	16°	9°	9		
XXXVII. G . . .	Wound of outer aspect of right leg, August 1914; lesion of external popliteal (complete R.D. followed in 18 months by partial R.D.), no reflex troubles, very moderate hypothermia.	Sound side	12	24°	8°	10		Feb. 1916
		Affected side	12	22°	8°	9		
XXXVIII. M . . .	Wound of right knee, September 1914; lesion of internal popliteal (plantar anæsthesia, complete R.D. of plantar muscles), hypothermia of foot, no other reflex disorders.	Sound side	13·5	20°	14°	11		Mar. 1916
		Affected side	14	18°	12°	10·5		

TABLE IV

Name.	Reflex Disorders Associated with Lesions of a Nerve Trunk (Upper Limb).		Systolic Pressure. Riva-Rocci-Vaquez.	Oscillations.		Gaertner.		Date of Examination.
				Brachial.	Radial.	Index.	Ring Finger.	
XXXIX. M . . .	Fracture of right carpus, May 1915; section of median at wrist (anæsthesia, complete R.D. of thenar eminence), cyanosis, pronounced hypothermia, exaggeration of mechanical contractility, paralysis of muscles of forearm and hand.	Sound side	12·5	18°	10°	9	14	April 1916
		Affected side	11·5	16°	6°	0	2	
XL. B . . .	Through and through wound of right elbow, September 1914; section of median (anæsthesia, complete R.D. of anterior muscles of forearm and thenar eminence); pronounced hypothermia, hypotonus, exaggeration of mechanical contractility of muscles of hand and posterior surface of forearm with paresis.	Sound side	13	21°	12°	9		Mar. 1916
		Affected side	12	18°	1°	5		

TABLE IV—(*continued*)

Name.	Reflex Disorders Associated with Lesions of a Nerve Trunk (Upper Limb).		Systolic Pressure. Riva-Rocci-Vaquez.	Oscillations.		Gaertner.		Date of Examination.
				Brachial.	Radial.	Index.	Ring Finger.	
XLI. C . . .	Through and through wound of right arm, June 1915; musculo-spiral paralysis (hypæsthesia, complete R.D. of supinator longus and posterior muscles of forearm), hypothermia, weakness of flexors, exaggeration of mechanical contractility of muscles of hand and anterior surface of forearm, with faradic hyperexcitability.	Sound side	12	20°	4°	11	14	Jan.
		Affected side	11	22°	2°	9·5	13	1916
XLII. B . . .	Through and through wound of right arm, April 1916; slight lesion of median (anæsthesia of index, with coldness of this finger, partial R.D. of thenar eminence), hypothermia, exaggeration of mechanical contractility of hand muscles.	Sound side	12	14°	6°	10	13	June 1916
		Affected side	12	12°	2°·5	5	10	

XLIII. B . . .	Wound of left arm, September 1914; musculo-spiral paralysis (complete R.D. of supinator longus and muscles of posterior surface of forearm), paresis of other muscles of forearm, hypothermia, exaggeration of mechanical contractility.	Sound side	13	20°	12°	10		Mar. 1916
		Affected side	13	18°	6°	7		
XLIV. D . . .	Through and through wound of left forearm, October 1915; lesion of median above wrist with complete R.D. of thenar eminence; reflex disorders in region of ulnar nerve.	Sound side	13·5	18°	8°	10	11	June 1916
		Affected side	13	18°	5°	6	10	
XLV. L . . .	Wound of right upper arm, June 1915; with complete R.D. and anæsthesia of inner surface of hand; hypothermia, exaggeration of mechanical contractility of forearm muscles, atrophy.	Sound side	11	20°	3°	8	12	Mar. 1916
		Affected side	11	20°	1°	11	10	
XLVI. V . . .	Right middle finger crushed, September 1914; painful neuritis of median (hypæsthesia, partial R.D. of thenar eminence), coldness of hand, especially index, exaggeration of mechanical contractility of muscles of hand.	Sound side	14	12°	5°	14	15	April 1916
		Affected side	14·5	16°	5°	10	15	

TABLE IV—(*continued*)

Name.	Reflex Disorders Associated with Lesions of a Nerve Trunk (Upper Limb).		Systolic Pressure Riva-Rocci-Vaquez.	Oscillations.		Gaertner.		Date of Examination.
				Brachial.	Radial.	Index.	Ring Finger.	
XLVII. C . . .	Wound of left forearm, November 1915; section of ulnar nerve, pronounced hypothermia, atrophy and exaggeration of mechanical contractility of forearm muscles.	Sound side	10·5	14°	5°	11		April 1916
		Affected side	11	7°	1°	9		
XLVIII. F . . .	Wound of right elbow, November 1914; musculo-spiral paralysis with complete R.D., reflex disorders in the area of the other nerves of the limb.	Sound side	13·5	14°	6°	6		April 1916
		Affected side	13	12°	5°	4		
XLIX. L . . .	Section of right forearm, April 1916; slight lesion of median (partial R.D. of thenar muscles), paresis of hand with increase of mechanical contractility of muscles of hand and forearm, temperature of hand very variable.	Sound side	12·2	14°	10°	12	12·5	July 1916
		Affected side	12·5	12°	6°	11	11	
L. E . . .	Wound of left elbow, August 1914; incomplete lesion of ulnar nerve (partial R.D.), hypothermia reaching to shoulder, most marked in 5th finger, atrophy and weakness of limb.	Sound side	12·5	20°	14°	13	12	June 1915
		Affected side	12·5	16°	16°	12	3	

LI. A . . .	Through and through wound of left forearm, June 1915; section of ulnar nerve, pronounced hypothermia, atrophy of forearm, exaggeration of mechanical contractility of thenar muscles.	Sound side	10·5	17°	5°	11	11	May 1916
		Affected side	10·5	14°	4°	9	1	
LII. L . . .	Through and through wound of right arm, August 1914; lesion of musculo-spiral and ulnar nerves (complete R.D.), hypothermia, paresis and increase of mechanical contractility of muscles innervated by median.	Sound side	13	20°	3°	10		April 1916
		Affected side	12·5	20°	1°·5	7		
LIII. F . . .	Through and through wound of right arm, December 1914; pseudarthrosis of humerus, lesion of musculo-spiral nerve (complete R.D.), marked hypothermia, exaggeration of mechanical contractility of hand muscles.	Sound side	12·5	22°	8°	12		April 1916
		Affected side	13	20°	4°	11		
LIV.	Cellulitis of right hand, April 1915; with lesion of median nerve (complete R.D.), retractions, hypothermia, spasms of hypothenar eminence, exaggeration of mechanical contractility of muscles innervated by ulnar.	Sound side	11·5	10°	7°	14	13	June 1916
		Affected side	11	16°	7°	9	7	

TABLE V

Name.	Reflex Disorders Associated with a Lesion of a Nerve Trunk (Lower Limb).		Systolic Pressure (Riva-Rocci-Vaquez) in dorsalis pedis.	Oscillations.		Gaertner. Second Toe.	Date of Examination.
				Femoral.	Tibial.		
LV. Le T . . .	Wound in left foot, October 1915; lesion of anterior tibial nerve at ankle (complete R.D. of extensor brevis digitorum); contracture of foot in equino-varus position, pronounced hypothermia, cyanosis, exaggeration of mechanical contractility of plantar muscles.	Sound side	12·5		20°	6	June 1916
		Affected side	12		9°	4	
LVI. B . . .	Wound of right thigh, October 1915; lesion of some branches of anterior crural (hypæsthesia and partial R.D. of vastus internus), reflex contracture of other portions of quadriceps, marked atrophy of thigh, pronounced hypothermia of thigh not extending to foot, exaggeration of mechanical contractility of muscles of leg and thigh.	Sound side	16·5	14°	12°	12	Dec. 1915
		Affected side	16·5	8°	8°	12	

LVII. F . . .	Through and through wound of left leg in lower third, May 1915; lesion of posterior tibial nerve with complete R.D. of plantar muscles, pain, plantar anæsthesia; atrophy, paresis, exaggerated mechanical excitability of muscles of leg and extensor brevis digitorum, hypothermia, exaggeration of left knee jerk.	Sound side	12·5		8°		Aug. 1915
		Affected side	12·5		5°		
		Sound side	13·2	18°	18°	9	July 1916
		Affected side	13	10°	9°	7	
LVIII. Ch . . .	Through and through wound of upper part of right thigh, September 1915; lesion of sciatic principally affecting fibres of external popliteal (complete R.D.); hypothermia, exaggeration of mechanical contractility of plantar muscles, exaggeration of knee jerk.	Sound side	12	28°	11°	11	July 1916
		Affected side	12	24°	6°	7	
LIX. L . . .	Wound of posterior aspect of left thigh, September 1914; complex section of sciatic (complete R.D. of all the muscles innervated by this nerve); atrophy and exaggeration of the mechanical contractility of the quadriceps, exaggeration of knee jerk, hypothermia.	Sound side	15	22°	10°	12	June 1915
		Affected side	15·2	12°	8°	5	

TABLE VI

Name.	Arterial Lesions, Associated with Lesions of Nerve Trunks and Reflex Disorders (Upper Limb).		Blood Pressure. Systolic Riva-Rocci-Vaquez.	Blood Pressure. Diastolic.	Oscillations. Brachial.	Oscillations. Radial.	Gaertner on Index.	Date of Examination.
LX. B . . .	Ligature of right axillary, July 1915; section of median and ulnar nerves (bullet wound), pronounced hypothermia, cyanosis.	Sound side	12·5	9	12°	8°	10	Nov. 1915
		Affected side	6	0	4°·	0°·5	1	
LXI. L . . .	Ligature of right brachial in upper third (bullet wound), June 1915; musculo-spiral paralysis (complete R.D.); hypothermia, 25° C. left hand and 20°·5 C. right hand, cyanosis, exaggeration of mechanical contractility.	Sound side	13	6·5	16°	10°	11	Jan. 1916
		Affected side	7·5	6·5	7°	1°·5	7	
LXII. B . . .	Ligature of left axillary for aneurysm, following wound, September 1914; complex lesion of brachial plexus, pronounced hypothermia, 26° C. right hand and 19° C. left hand.	Sound side	13	8·7	18°	9°	9	Feb. 1916
		Affected side	9·5	9	5°	1°	5	
LXIII. G . . .	Ligature of left axillary, October 1915 (bullet wound), lesion of musculo-spiral, ulnar and median; very slight	Sound side	13	10	14°	6°	13	Dec. 1915
		Affected side	0	0	6°	3°	6	

	hypothermia, 29° C. right hand and 27° C left hand.							
LXIV. P . . .	Obliteration of right brachial, following bullet wound, May 1915, found on operation; paralysis of the whole brachial plexus; hypothermia, 27° C. left hand and 23° C. right hand, pronounced cyanosis.	Sound side	11	9·5	8°	5°	10	Dec. 1916
		Affected side	0	0	0	0	0	
LXV. S . . .	Forceps left 4 days on right brachial, September 1914; lesion of ulnar nerve, hypothermia, cyanosis, exaggeration of mechanical contractility.	Sound side	11·5	7·5	22°	6°	8	Feb. 1916
		Affected side	7·5	7·5	12°	1°	8	
LXVI. Cl . . .	Obliteration of left axillary artery following bullet wound, October 1914; lesion of median and ulnar nerves, hypothermia, 27° C. right hand and 15° C. left hand, pronounced cyanosis.	Sound side	15	9	20°	12°	12	Dec. 1915
		Affected side	0	9	3°	1°	10	
LXVII. B . . .	Bullet wound of right arm, September 1914; obliteration of brachial by tourniquet; lesion of median, hypothermia, 25° C. left hand and 17° C. right hand, cyanosis, exaggeration of mechanical contractility.	Sound side	12	7	26°	10°	7	Dec. 1915
		Affected side	8	8	12°	1°·5	6	

TABLE VII

Name.	Arterial Lesions with Association of Lesions of Nerve Trunks and Reflex Disorders.		Blood Pressure. Systolic Riva-Bocci-Vaquez.	Blood Pressure. Diastolic.	Oscillations. Femoral.	Oscillations. Tibial.	Gaertner on Index.	Date of Examination.
LXVIII. S . . .	Section by bullet of right popliteal, May 1915; tourniquet, sloughing of toes; lesion of external popliteal nerve, foot red and hot, hypothermia of calf.	Sound side	15·5	8·5		16°		Feb. 1916
		Affected side	0	0		0°·5		
LXIX. M . . .	Ligature of left femoral, October 1914, for aneurysm following wound, slight lesion of external popliteal, *absence of any reflex disorder*, no hypothermia, nor exaggeration of mechanical contractility.	Sound side	14·5	9		8°		April 1916
		Affected side	13	10·5		2°		
LXX. C . . .	Ligature of right femoral in upper third, May 1915; *no nervous lesions;* very slight hypothermia, no exaggeration of mechanical contractility.	Sound side	15	10·5	16°	8°		Mar. 1916
		Affected side	0	0	2°	1°		

LXXI. V . . .	Ligature of right femoral in upper third for wound, January 1916; *no nervous lesions*, pronounced hypothermia, cyanosis, exaggeration of mechanical contractility.	Sound side	20	11	32°	10°		July 1916
		Affected side	0	0	6°	0		
LXXII. C . . .	Obliteration of left femoral in lower third with diffuse aneurysm from bullet, May 1915; *no nervous lesions*, pronounced hypothermia, 24° C. right foot and 20° C. left foot, exaggeration and slowness of mechanical contraction of muscles.	Sound side	14·5	9·5	14°	3°		Nov. 1915
		Affected side	12	11	12°	0°·5		

Influence of Hot or Cold Baths on the State of the Local Circulation

In the normal subject the limb when plunged into water at 40° C. for five minutes becomes red, its temperature rises from 23°–27° C., the arterial oscillations become amplified and the arterio-capillary pressure, as taken by Gaertner's tonometer, rises. In a cold bath (12° C.) of the same duration the temperature falls, and sinks, for instance, from 25°–15° C., or from 18°–12°·5 C., while the amplitude of the oscillations and the arterio-capillary pressure diminish. But as a general rule all these changes disappear fairly rapidly, and in about ten minutes the limb has returned to its former condition.

In the subject suffering from *reflex nervous disorders* the temperature, as the result of the hot bath, frequently rises on the affected side more than on the sound side. Under the action of the cold bath the fall of the local temperature is more marked in the affected limb. Thus, in one of our patients (Table I, Case II), we noted—

	Before the bath.	After bath at 40° C.
Sound side . . .	23°	27°
Affected side . . .	15°	28°

	Before the bath.	After bath at 12° C.
Sound side . . .	18°	12°·5
Affected side . . .	14°	9°

In another patient we noted—

	Before the bath.	After bath at 12° C.
Sound side . .	25°	15°
Affected side . . .	27°	13°

We may add that the return to the original condition is generally slower in the affected limb than in the sound limb.

As regards *the state of the circulation*, this is modified in a similar manner in that as the result of a warm bath the amplitude of the oscillations tends to increase on the

affected side more than on the sound side. It is the same as regards the arterio-capillary pressure. Here are some examples—

CASE II (TABLE I)

	Before the bath.		After bath at 40° C.	
	Radial oscillations.	Gaertner.	Radial oscillations.	Gaertner.
Sound side . .	7	9	16	10
Affected side. .	1	0	4	9

At the same time the skin of the affected side becomes hot and the muscular contraction on percussion becomes perceptibly more rapid and less ample.

CASE IV (TABLE I)

	Before the bath.		After bath for 5 min. at 40° C.	
	Radial oscillations.	Gaertner.	Radial oscillations.	Gaertner.
Sound side . .	4	10	6	11
Affected side . .	2	11	5	12

CASE VIII (TABLE I)

	Before the bath.		After bath at 40° C.	
	Radial oscillations.	Gaertner.	Radial oscillations.	Gaertner.
Sound side . .	5	7·5	8	10
Affected side . .	3	4	8	8

Another day—

Sound side . .	4	4
Affected side . .	2	8

In both experiments the affected hand had become definitely hotter than the sound hand, and the mechanical excitability of the muscles, which had been very exaggerated, had returned to normal.

Case IX (Table 1)

	Before the bath. Radial oscillations.	After bath at 40° C. Radial oscillations.
Sound side	4	12
Affected side . . .	2	22
Fresh experiments a few days later—		
Sound side	5	18
Affected side . . .	6	22

It should be noted that the hyperthermia in the affected limb can still be found thirty minutes after the bath. This case is a very striking one, as it clearly shows the disturbance of the vaso-motor mechanism in the affected limb.

Case XV (Table II)

	Before the bath. Tibial oscillations.	After a bath of 5 minutes at 40° C. Tibial oscillations.	After a bath of 10 minutes at 40° C.
Sound side .	8	8	14
Affected side .	1	6	12

Simultaneously with a progressive increase in the oscillation a full pulse was felt in the dorsalis pedis artery, which could not be felt on the affected side before the bath when the foot was very cold. The cyanosis and œdema persisted, but the mechanical excitability, which had been very much increased before the bath, became normal again.

Case XVI (Table II)

	Before the bath. Tibial oscillations.	After bath at 40° C. Tibial oscillations.
Sound side . . .	11	12
Affected side . . .	6	13

Case XXVI (Table II)

	Before the bath. Tibial oscillations.	After bath at 40° C. Tibial oscillations.	15 min. after bath. Tibial oscillations.
Sound side .	6	6	6
Affected side .	2	8	8

Cold baths produced the opposite effect; the amplitude of the oscillations diminished in the affected limb more than in the sound limb. Thus in one of the cases observed we noted—

	Before the bath. Radial oscillations.	After bath at 12° C. Radial oscillations.
Sound side . . .	3	0·75
Affected side . . .	3	0

In another patient we noted—

	Before the bath.		After the bath.	
	Radial oscillations.	Gaertner.	Radial oscillations.	Gaertner.
Sound side .	4	12	3	12
Affected side .	4	6	2	4

We sometimes employed this method during the summer months to demonstrate the circulatory disturbances in the paretic or contractured limb in doubtful cases.

In patients presenting *reflex nervous disorders associated with a lesion of a nerve trunk*, as in the preceding case (pure reflex disorders), the hot bath causes the amplitude of the oscillations to increase more on the affected side than on the sound side; it is the same for the arterio-capillary pressure. The following are two examples—

Case XLIII (Table IV)

(Musculo-spiral paralysis with pronounced reflex disorders.)

	Before the bath.		After bath at 40° C.	
	Radial oscillations.	Gaertner on the index.	Radial oscillations.	Gaertner on the index.
Sound side	12	10	14	10
Affected side	6	7	16	7

Simultaneously with a considerable increase in the oscillations on the paralysed side, the hand which had been cold before the bath became as hot as on the sound side, the white patch disappeared equally rapidly on the two sides, and the mechanical excitability of the hand muscles perceptibly diminished.

Case XLIV (Table IV)

(Paralysis of the median with reflex disorders.)

This case is still more striking because it shows the rise of arterio-capillary pressure in the index, due to the bath, in a case of lesion of the median.

	Before the bath.		
	Radial oscillations.	Gaertner on the index.	Gaertner on the ring finger.
Sound side . .	8	10	11
Affected side . .	5	6	10

	After bath at 40° C.		
	Radial oscillations.	Gaertner on the index.	Gaertner on the ring finger.
Sound side . .	10	11·5	14
Affected side . .	10	10·5	15

In the case of *arterial obliteration* the local temperature rose under the action of the warm bath, but less than in cases of reflex disorders; the arterio-capillary pressure also rose, but the oscillations were not modified or showed only a very slight increase, *e. g.*, if the amplitude was 0·5 before the bath, after the bath it was 0·5 or 1° (143) (161).

To sum up, we have studied the state of the peripheral circulation in the following conditions—

1. In reflex nervous disorders unaccompanied by lesions of the nerve trunks or large vessels (pure reflex disorders).
2. In lesions of the nerve trunks unaccompanied by reflex nervous disorders, with the motor disorders exclusively localised in the area of the region affected.
3. In lesions of the nerve trunks associated with reflex nervous disorders.
4. In lesions of the large vessels either alone or combined with various nervous associations (lesions of the nerve trunks, reflex nervous disorders).

How are these various conditions to be distinguished from one another?

We have described above the condition of the circulation in cases of *pure reflex nervous disorders*, of which the following are the essential features—

(*a*) Diminution of the amplitude of the oscillations, ill-marked in the brachial and femoral, and often pronounced in the radial and tibial arteries.

(*b*) Fall of the arterio-capillary pressure, as measured by Gaertner's instrument.

(*c*) Absence of any change in systolic or diastolic arterial pressure.

(*d*) Considerable increase in the affected limb as the result of the application of heat, of the amplitude of the oscillations, which may become equal or superior to that of the oscillations in the sound limb.

In *pure lesions of the nerve trunks* there may not be any appreciable change in the circulation.

When *reflex nervous disorders are associated with a lesion of a nerve trunk*, the state of the circulation resembles that seen in pure reflex disorders, except that the vaso-motor phenomena generally predominate in the area of the affected nerve, as can be ascertained by Gaertner's apparatus; the fall of the arterio-capillary pressure, as well as the hypothermia and cyanosis are more marked in the index, when the median nerve is affected, and in the ring and little finger in lesions of the ulnar; after application of heat, the difference diminishes or disappears.

In cases of pure or associated *arterial obliteration*, the amplitude of the oscillations is considerably diminished, and in this respect this class of cases resembles reflex nervous disorders; but contrary to what is seen in the latter, the systolic blood pressure is very much weakened when the artery is obliterated, and another important distinguishing feature is the fact that the application of heat does not increase the amplitude of the oscillations or only in an insignificant degree.

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SECTION IV

THE PSYCHONEUROSES OF WAR

THE
PSYCHONEUROSES OF WAR

Chapter I

ELEMENTARY PSYCHOMOTOR COMPLAINTS

Psychoneurotic motor symptoms, paralyses or contractures, have been in evidence amongst soldiers since the very commencement of the war. Several cases a day have been found both in hospitals near the battle-front and in the general hospitals. They are undeniably the most common and therefore the most important of the neuroses of war.

These motor disorders take the form of a flaccid paralysis or paralysis with contracture, when they more or less closely resemble the true organic paralyses, either flaccid or spasmodic ; thus we find hemiplegias, paraplegias, and monoplegias. Sometimes, as in certain contractures of the hands, feet, or trunk, they defy any central or peripheral classification and may assume the most infinite variations in type.

§ 1. Paralyses and Contractures of One or More than One Limb

hemiplegia, monoplegia, paraplegia

In spite of their diversity, the psycho-neuropathic motor complaints described under the above heading have certain common characteristics : they appear under the same etiological conditions, they exhibit specific clinical and " pathognomonic " signs which permit of their differentiation from organic diseases and, lastly, they react to therapeutic measures in a

similar manner. Therefore we can study their etiology and differential diagnosis together, only their clinical aspects needing separate treatment, always bearing in mind the fact that the various types often overlap each other.

Etiology.—The conditions underlying the development of a hemiplegia, a monoplegia of an arm or leg, or of a paraplegia, are practically identical.

Sometimes they originate from some fairly severe trauma not entailing any grave wound, a cerebro-spinal concussion from the explosion of a projectile or a mine close at hand but without any obvious bodily wound, shock from the collapse of a trench, etc. In other cases the onset of the paralysis remains obscure and functional phenomena develop without any mental or physical shock. It is not exceptional to find that these paralyses or contractures are preceded by more or less vague pains in the joints of the limbs which have been contused. They are often intractable and are the basis of many cases of paralysis which are apparently of spontaneous development. Lastly, these motor affections may develop in hospital during the course of an infectious disease contracted in the trenches.

The situation of the trauma has no bearing on the site of development of the paralysis. A wound of the shoulder or of the hand may be equally effective in producing absolute impotence of the upper extremity. A scar, proving the existence of a real initial trauma, may even be found at a great distance from the affected limb. Further, the severity of the wound bears no relation whatever to that of the paralysis; on the contrary, it appears that in most cases it is the most insignificant wound that brings about the most serious functional paralysis.

The *Onset* of these motor affections is much the same, whatever form they may take. Sometimes they appear immediately after the trauma, as soon as the patient recovers from the emotional shock that he has just experienced (*e.g.* having been buried

in earth or débris, or having sustained some local trauma); or, if he has lost consciousness, at the moment he regains it in the field-ambulance or in any of the Red Cross departments. Often the hemiplegia, monoplegia, or paraplegia may not become manifest until several days later, and during this period — the *phase de méditation* of Charcot — the soldier only exhibits vague symptoms in keeping with a physical and psychical disorder occasioned by the trauma: more or less diffuse aches and pains, symptoms of vertigo following a labyrinthine concussion (Lhermitte), and general asthenia, the result of repeated fatigue and of shock, due to cerebro-spinal concussion or emotion.

Clinical Aspect.—We shall study successively the principal clinical types of paralysis.

Hysterical Hemiplegia.—Although a good deal rarer than paraplegia or the monoplegias of the same nature, the so-called "hysterical" hemiplegia, or better hysterical hemiparesis, is far from being a rarity. However, it can be said that the war has not added a large number of cases. Indeed, if only those directly referable to physical or psychical trauma in the course of the war are considered, their number is strictly limited.

Hysterical hemiplegia is rare in consequence of a wound of a limb; this is conceivable when one considers that the local trauma limits its action to fixing upon a restricted region of the body the functional disturbance (bound up with the emotion and the representation of the emotion). Cases are still rarer in which the hemiparesis appears as a result of a wound of the head, which, though slight, is upon that region of the skull which lies over the motor centres for the limbs which exhibit the paresis or paralysis. We shall return to such cases later.

The features of so-called hysterical hemiplegia are too well known to need description, all the details observed during the war being found in the classical type.

PLATE I.

Fig. 1. Hysterical Hemiplegia of the Left Side. T. M., billet-officer, —— cuirassiers. Villejuif Hospital, 25th January 1915.

Onset of loss of power in the left side in November 1914, following on fatigue and emotion. Complete paralysis of the left arm and paresis of the left leg. Anæsthesia (of the hysterical type) of the left arm and of the left leg as far as the middle of the thigh. Dragging gait. Plantar reflexes flexor on both sides.

Recovered in six months, able to rejoin his regiment and ride a horse.

In his past history there was a record of a similar affection lasting a month at the age of 16½.

Fig. 2. Monoplegia of the Right Leg : Pseudo-coxalgic Type. M. A., —— infantry regiment. Villejuif Hospital, 9th February 1915.

Wounded on the 9th September 1914. Wound of the right thigh. All active movements were made equally well on the right and left sides. Power of the right limb slightly diminished, especially that of extending the knee. Reflexes normal. Slight lameness : toes turned outwards. The sole of the foot lay quite flat on the ground. On admission, the patient exhibited absolute and complete anæsthesia of the whole limb, extending up to the umbilicus ; this anæsthesia disappeared on the day of admission after treatment with a powerful faradic current applied to the skin.

Claudication completely cured in a month by re-education and electrical treatment.

Figs. 3 and 4. Psycho-neuropathic Astasia-Abasia, following Concussion due to the Bursting of a Shell. B., aged 25.

The patient could only walk with his eyes fixed on the ground. Walking interrupted by movements of latero-pulsion. Tight-rope walker's gait.

Fig. 5. Spasmodic Neuropathic Paraplegia, following Concussion due to a Shell. A., aged 33. Neurological Centre of District VIII.

Normal cutaneous and tendon reflexes. The patient takes long strides and holds the body absolutely rigid.

FIG. 1.—Hysterical hemiplegia of the left side.

FIG. 2.—Hysterical monoplegia of the right leg.

FIGS. 3 and 4.—Psycho-neuropathic Astasia-abasia.

FIG. 5.—Neuropathic spasmodic paraplegia.

It is known that hysterical hemiplegia occasionally takes the form of an absolute motor paralysis of both the upper and lower limb. Walking is possible with the aid of a stick or crutches. The dragging gait is quite characteristic; the point of the foot drags along the ground, the trunk is bent, and at each step its weight is thrown on to the limb affected by paresis.

With regard to the upper extremity, it is very often more affected than the lower; hanging beside the body, it is incapable of any voluntary movement. Neither the face nor the tongue share in the paralysis, and the apparent facial paralysis sometimes met with is really due to a tonic glosso-labial spasm of the opposite side. This spasm shows itself not only by a permanent contracture of the muscles of the face and tongue on the side opposite to the paralysis, but also by clonic twitching of the contracted muscles.

The cause of this distortion of the features is further proved by the fact that in involuntary and automatic imitative movements, the two sides of the face contract with equal energy and all asymmetry disappears.

The cutaneous and tendon reflexes are normal and muscular tone is equal on the two sides. This rule, however, has certain exceptions which it would be dangerous to ignore. Although the neurosis in itself may be incapable of modifying the normal cutaneous and tendon reflexes, this is not to say that the latter are incapable of modification in a purely neuropathic hemiplegia of old standing. Following on prolonged functional inactivity, the muscles of the paralysed limb undergo atrophy, and this itself necessitates an inequality of the tendon and bone reflexes; the latter will be more marked on the side of the hemiplegia, without always being accompanied by true ankle clonus or patellar clonus. We have here an example of a common law that simple muscular atrophy secondary to immobility or of a reflex nature

(arthopathies, fractures) is accompanied by increased tendon and bone reflexes.

Thus the discovery of such inequality of reflexes should not, *ipso facto,* entail the diagnosis of organic hemiplegia when the paralysis is accompanied by amyotrophy.

The plantar, cremasteric and abdominal reflexes remain normal in the majority of cases, although abnormalities have been observed (Dejerine, Sollier, Paulian, Lhermitte). But in such cases the hemiplegia is always accompanied by a significant cutaneous anæsthesia, and the absence of cutaneous reflexes is partly due to the latter. The normal plantar reflex is never reversed, and extension of the hallux definitely excludes the existence of a functional paralysis.

Hysterical hemiplegia is frequently associated with disturbances of sensation, to which we shall return later (the so-called hysterical hemianæsthesia).

During the last few years, in France and in other countries, there has been described a series of signs and symptoms for the differential diagnosis of hysterical and organic hemiplegia. One of the authors (Lhermitte) has proposed their grouping under the title of *The minor indications of organic hemiplegia*: (*a*) Hyperextension of the forearm or the arm due to muscular hypotonia ; (*b*) platysma sign (absence of contraction of this muscle on the paralysed side ; (*c*) associated movement of flexion of the thigh and pelvis (spontaneous flexion of the thigh on the pelvis when the patient in the sitting posture throws himself smartly backwards into the dorsal position) (Babinski) ; (*d*) sign of " complementary opposition " (Hoover) : when the patient is asked to raise the paralysed limb above the plane of the bed, he carries out the reverse movement of the healthy limb, which is forcibly pressed against the mattress ; (*e*) Heilbronner's sign : the muscular hypotonicity is shown by increase in the transverse diameter of the thigh ; (*f*) flexion of the toes on gently percussing the sole

of the foot (Rossolimo); (*g*) flexion of the small toes on percussion by a hammer of the dorsum of the cuboid bone (Mendel-Bechterew); (*h*) extension of the great toe on deep friction of the calf muscles (Oppenheim) or by pinching the Tendo Achillis (Schäffer's or Gordon's sign); (*i*) lastly, transverse pressure on the tarsus or forcible flexion of the toes produces retraction of the lower limb, even when the latter is quite incapable of any voluntary movement (contraction sign of Pierre Marie and Foix).

All these signs are wanting in "hysterical" hemiplegia.

Hysterical hemiplegia with contracture or, more correctly, *hysterical hemi-contracture* develops suddenly: the limbs appear to exhibit an abrupt rigidity or complete contraction.

The condition of hypertonicity does not occur after a phase of paralysis, but spontaneously: this is the reverse of the case in spastic organic hemiplegia.

This hemi-contracture develops in two forms:—

(1) One characterized by an intense contracture of the upper and lower limb to such a degree that all passive movement is impossible. The lower limb is extended, the foot in equino-varus position and absolutely rigid. The upper extremity assumes a position of flexion or extension, but the flexed type is more often seen.

(2) The other, in which the hemiplegia assumes both a spastic and flaccid type: spastic as regards the contracted lower extremity, flaccid as regards the upper, which hangs absolutely inert beside the body. The contracture of the lower limb, possibly very marked, may be of the flexed or the extended type. In this condition the face is usually quite unaffected.

Henri Claude and R. Porak have recently made an analysis of movement in this paresis (hysterical or psychoneurotic "functional inactivity") by means of the ergograph.

In hysteria the simplest movement—*e.g.* flexion

of the second finger—is badly performed and incoordinate. With a submaximal weight the ascending part of the curve is irregular; made up of a series of sudden jerks, it gives the impression of an uncontrolled nervous influx which is suddenly discharged alternately in the synergic and antagonistic muscles concerned in the movement. The summit of the tracing is irregular, slow, and protracted; the descending line is equally irregular, interrupted by notches representing muscular discharges at the moment of relaxation.

If one studies a protracted voluntary contraction or, in other terms, if we examine the graphic record of static muscular work, it is found that in the hysterical, neuropathic subject the tracing is diminished in height, much elongated if the work demanded is minimal, and exhibits irregularities at the moment of relaxation.

Paralytic and spastic phenomena and disorders of sensation do not make up a complete picture of functional hemiplegia; to these must be added trophic and vasomotor manifestations and changes in the electrical reactions of nerve and of muscle, the nature of which must be understood.

The old conception of hysteria admitted without dispute the existence of these muscular, cutaneous, and tendon symptoms, which Babinski has justly ascribed to their real cause: malingering or fraud.

In long-standing hysterical hemiplegia, as, indeed, in any paralysis that has lasted sufficiently long, we find:—in the muscular system, diffuse amyotrophies without Reaction of Degeneration; in the osseous system, decalcification and rarefaction of the cancellous bone; lastly, from the electrical point of view, the paralysed muscles and nerves show a diminished reaction to faradic and galvanic stimuli after a certain length of time; the muscle-jerks remain brisk.

Rumpf has cited recently the production of a

fibrillary tremor, at first isolated and limited to the muscle-group directly excited, as characteristic of the contraction seen in hystero-traumatic hemiplegia. It is a muscular tremor reminiscent of a cornfield blowing in the breeze. Cooling the limb by applying ice is equally effective in determining this phenomenon. But Rumpf's sign is by no means constant in hysterical hemiplegia, and, on the other hand, it can often be observed in the organic variety (Pierre Marie). Lastly, as artificial cooling of the paralysed limb or muscle is sometimes effective in its production, it is legitimate to assume that the frequent low temperature of the paralysed limb is alone sufficient to bring about muscular tremor after fatigue.

With regard to vasomotor symptoms—*lowering of temperature, cyanosis of the paralysed extremities, profuse sweating, œdema*, etc.—these are also the result of functional inactivity. This is proved by their rapid disappearance when the hysterical paralysis or contracture has been cured by psychotherapy. We shall return to this subject, however, in a later section.

In any case, vasomotor or trophic phenomena cannot be considered as manifestations of a psycho-neuropathic order but rather as organic affections due to immobilization.

With regard to segmental œdema, which has been seen by all neurologists during the course of the war, this should immediately suggest malingering and entail a search for marks of a constriction. Moreover, these " constriction œdemas " as Sicard rightly terms them, do not occur in hysterical hemiplegia, but rather in cases of pure brachial monoplegia or in wounded men who have an almost intact mobility.

Hysterical Monoplegia and Paraplegia

Motor affections localized in the upper or lower limbs—*monoplegia of the upper or lower extremity* or

of both lower extremities, *paraplegia*—are more frequent than psychoneurotic manifestations of the hemiplegic type. The number of these cases observed during the war is large, and the diagnosis and treatment of these psychical paralyses so often comes under medical observation that we must devote special attention to them.

Brachial Monoplegia.—There are two types, flaccid and spastic, the former being the more common.

(1) In the *flaccid type* the paralysis is complete and absolute; no active movement is possible; the upper extremity hangs down and swings as an inert body beside the trunk when the patient moves.

The shoulder is lowered, the forearm in passive extension, the hand also extended with outstretched and adducted fingers. The patient is incapable of carrying out the simplest movement and when by its own weight the immobile limb takes up an awkward posture, he replaces it in a more favourable position by means of the healthy member.

If the patient is sternly commanded to perform some elementary movement or other, and does not succeed, the limb remains inert; but sometimes a muscular tremor or undulation is observable under the skin, often in muscles which should not logically share in the movement in question. This is of some importance in diagnosis.

(2) In the *type with contracture*, rarer than the flaccid form, the limb is fixed, either in flexion or extension. The muscles are strongly contracted, projecting under the skin, hard to the touch. No movement is possible either active or passive, except movements at the shoulder-joint, which may be preserved. In contrast with organic contractures, all efforts to mobilize the limb are not only ineffectual but exaggerate the intensity of the contracture. The same holds for the application of Esmarck's bandage, which, as Brissaud has shown, causes re-

laxation of organic but exaggeration of inorganic contractures.

Crural Monoplegia.—According to our experience, this is much less often observed than brachial monoplegia.

(1) The *flaccid form* is especially rare and hardly ever complete; the patient is always able to execute some voluntary movements and to walk with the help of crutches or sticks.

It should be noticed that during the automatic movement of walking, muscles seem to contract which are absolutely inert when their mobility is tested with the patient lying down; on the other hand, this is very exceptional in an organic monoplegia.

(2) The *form with contracture* is common. It represents one of the most frequent types of the neuroses of war.

(*a*) In the *flexed type* the leg is incompletely flexed on the thigh and the thigh on the pelvis; contact with the ground is only made by the ball of the great toe. Walking is laborious and slow and accompanied by a limp. All the contracted muscles are hard to the touch and stand out in striking relief under the skin. Both active and passive movements are extremely limited, and as a rule it is impossible, even by employing considerable force, to correct the vicious posture.

(*b*) In the *extended type* the lower limb is absolutely straight, all the segments are extended, including the foot, which, as in the last condition, only reaches the ground by the ball of the toe. The result is that walking is only possible by tilting the pelvis on the affected side, this being necessarily accompanied by a curvature of the vertebral column with the convexity directed to the healthy side. Sometimes the foot is not contracted in extension, but immobilized in a position closely resembling the normal, only slightly extended. In such conditions walking is

much easier, and the contracture is not accompanied by any deviation of the vertebral column.

All the forms of contracture we have just described will, if allowed, be complicated by muscular shortening. Thus Charcot especially urged that hysterical contractures should not be allowed to get out of hand.

Paraplegia.—Of all the elementary motor neuroses of war, the paraplegic syndrome is the most commonplace and possibly the most frequent form. We shall not discuss here the various affections of the gait, which are dealt with in a later section (p. 751), but confine ourselves to true paraplegia without any added symptoms.

(1) In the *flaccid form*, which is the commonest type, the lower limbs lie extended on the bed; in spite of all efforts, the individual is incapable not only of moving them but also of producing even feeble contraction in any muscle. It is only by means of his arms that the patient can move the paralysed limbs, which are absolutely inert. All passive movements can be made with the greatest ease. Walking is quite often possible with the help of crutches. In the latter case it is important to note carefully the mobility of the lower limb during walking, as this gives us interesting information. It is seen that at the time of forward progression the lower limbs execute regularly the necessary movements, though with little force; and that certain muscles, incapable of voluntary contraction when the patient is in the recumbent position, contract fairly energetically during the automatic act of walking.

(2) *Paraplegia with Contracture* only assumes one type: that of extension. The two limbs are adducted and drawn together with the knees touching or slightly crossed, as in infantile spastic paraplegia (Little's syndrome); the feet participate in the general extension, and are in the equino-varus position. Attempts at passive movement only accentuate the contractures. The sacro-lumbar muscles are

often equally contracted, projecting on each side of the vertebral column; in such cases the lumbar part of the spine is like a rigid bar, the trunk following movement of the lower limbs.

As often happens in psychoneurotic paralyses, paradoxical cases are found, in which the paraplegia is, properly speaking, neither spastic nor flaccid but a combined spastic-flaccid type. In the recumbent position the patient is able to execute certain simple movements of the lower limbs; the latter are supple, sometimes even slightly hypotonic, but when he is placed upright by means of two sticks, his legs stiffen, the muscles stand out under the skin and he progresses with the lower extremities in complete extension, without any flexion of the knee.

The concurrence of organic and functional disorders is extremely frequent, and the resulting paraplegia, which at first sight appears entirely functional, is really made up of two kinds of phenomena, organic and neuropathic. This is sometimes seen in disturbances of the spinal medulla of long standing.

At its onset the paraplegia corresponds with a medullary lesion, shown by the classical and characteristic signs; then, when these have disappeared, the paralysis remains unchanged or is occasionally even increased. Apart from a certain liveliness of the tendon reflexes, it is impossible to discover any organic manifestation, but the impotence remains absolute. There is complete anæsthesia of the lower limbs, reaching to the sub-umbilical plane. Walking is difficult, and when undertaken the lower limbs are seized by marked contractures. We have even observed cases in which the patient progresses by hopping, and a false patellar or ankle clonus has occurred spontaneously or after manipulation.

Lastly, there are rare cases in which the paraplegia with contracture is organic in one limb and neuropathic in the other, and the individual presents the series of spastic signs on the one side and false clonus and hysterical tremor on the other.

The **Prognosis** of the monoplegias, like that of the functional hemiplegias, varies according to the clinical forms which they assume and according to whether they are free from any organic element.

All the pure forms should become completely cured, if given correct treatment. The neuropathic paraplegias are, however, especially liable to relapse.

Differential Diagnosis.—We must first consider the chief characteristics which specially belong to the paralyses of psychical order and which permit of their differentiation from organic affections.

In making this diagnosis, importance must be attached to the following points: the conditions of their incidence, the clinical forms in which motor neuropathic affections appear, careful inquiry for and absence of somatic signs, psychological study of the individual, the course of the affection and the results of treatment.

(1) *Motor neuropathic affections* are, for the most part, directly referable to some trauma suffered at the front. They appear either immediately after the concussion or emotional shock, or after a "phase of contemplation" lasting some days.

(2) *From the clinical aspect,* functional paralyses are distinguished by more obvious and blatant phenomena than organic motor affections: a flaccid paralysis is always more absolute; contracture is spontaneous, not preceded by flaccid paralysis, always very pronounced and causing extreme deformity. These contractures show many variations, are accompanied by a complete loss of muscular elasticity and do not yield to Esmarck's bandage.

(3) *Associated Signs*, such as pain and objective disorders of sensation, are rarely absent.

(4) During *Examination of the Paralysed Limb or Limbs*, when contractures are present, the patient's attempts to prevent movement of the immobilized joints should be carefully noted; contraction of the antagonistic muscles, even, may often be observed.

(5) *Complete absence of any change in the skin or tendon reflexes, of ankle clonus, of vasomotor, trophic, or sympathetic symptoms, of muscular atrophy and of change in the electrical reactions* (except in old cases with long-standing immobilization), are all fundamental signs of these conditions.

(6) Lastly, a good deal of importance is attached to *a psychological study of the patient*, such as the way in which he tells of his misfortune, the importance he attaches to the affection he is suffering from or believes himself to be suffering from, the lack of desire or hope of cure which he displays, and, finally, the manner in which he reacts to therapeutic measures (resistance, nervous crisis, tremor, outburst of weeping, etc.).

It would be out of place in this connection to review all the organic affections which might give rise to difficulty. We shall confine ourselves to citing a few of the more recent observations which we have drawn from a neurological study of the war.

In **Hemiplegia** differential diagnosis is easy, owing to the numerous signs which separate the functional from the organic variety. In actual practice it is especially the cases of mixed hysterical and organic origin, of psychical hemiparesis developing as a result of some cranial trauma, which give rise to difficulties.

We may cite the case of a man with a gun-shot wound in an " indifferent " region of the skull (over the frontal or the right occipital lobe). He exhibits complete flaccid hemiplegia, without any apparent reflex changes ; the face is not affected ; the diagnosis of hysterical hemiplegia seems obvious, the very cause of the complaint seeming to warrant this interpretation ; and under the influence of suggestive treatment the paralytic phenomena quickly subside, but complete cure is not obtained. The individual is put down as a malingerer or exaggerater, and is sent back to his regiment with a sharp reprimand. After an interval the man is returned to a neurological centre for a fresh examination. Then it is that

minute signs of organic trouble are discovered: the spreading out of the toes (*signe de l'éventail*), Oppenheim's sign, the Mendel-Bechterew reflex, the diminution of the abdominal and cremasteric cutaneous reflexes on the hemiplegic side, sensation remaining normal. These are sufficient to justify the diagnosis of some organic lesion of the pyramidal tract.

In another case we may find a wounded man with a brachial monoplegia, accompanied by paralysis of the lower limb on the same side. But whilst changes in the reflexes and in reaction to electrical stimuli (*e.g.* in lesions of the radial nerve) are found in the upper extremity as evidence of the organic origin of the paralysis, nothing of this kind can be demonstrated in the lower extremity. The phenomenon of complementary opposition is absent. There can be no question of the correct diagnosis: there is a hystero-organic association, the paralysis of the upper limb being organic and that of the lower purely functional.

The same holds good in the case of the **monoplegias and paraplegias.** Certain wounds of the skull in the parietal region may be followed by a flaccid or, more often, a spastic monoplegia of the leg of a psychical order, which may be mistaken for an organic monoplegia by the less-skilled observer; this the more since the injury was in a portion of the skull covering the cortical motor centre for the limb in question and since, perhaps, additional evidence is adduced from some slight depression of the external table. But in contrast with organic monoplegia, the hysterical contracture is *immediately* established, is extremely pronounced and results in complete deformity of the foot, or is accompanied by external rotation of the entire limb. The cutaneous and tendon reflexes are normal and there is no disturbance of sensation as in cerebral lesions.

It may appear unnecessary to discuss the differential diagnosis of hemiplegia with contracture and tetanus.

But there are certain forms of tetanus infection, chronic and limited, in which mistakes may be made

Cases in which there is legitimate cause for doubt are those that come under the heading of hysterical hemi-tetanus (Boker) on account of the exaggeration of the tendon and bone reflexes always found in chronic tetanus (Claude and Lhermitte), and which is very often accompanied by true ankle clonus and patellar clonus and by the spreading out of the toes (*signe de l'éventail*). However, the discovery of even a minor degree of trismus, of contraction of the abdominal muscles, of electrical hypersusceptibility of nerve and muscle (Claude and Lhermitte) will clinch the diagnosis of tetanus.

In the last place we must mention the so-called *habit-paralyses* and *habit-contractures,* which certain authors have distinguished from the hysterical motor affections. To our way of thinking, it is only a difference of degree, often difficult to fix. Whether a crural or brachial monoplegia or a functional paraplegia has arisen spontaneously without any previous organic lesion, or whether, on the other hand, it has followed an organic lesion—neuritis of the brachial plexus, sciatic neuritis, or discrete lesion of the cauda equina—the motor impotence caused by prolongation or persistence of the symptoms points in either case to a disorder or defect in the psycho-motor mechanism. They are individuals who, more or less unconsciously, have continued not to make any use of a limb, to persist in some vicious posture, to continue limping in spite of the complete disappearance of any organic lesion. The habit-claudication, described later in dealing with affections of the gait, belongs to this class.

§ 2. Paralyses and Contractures limited to one Anatomical Region or Group of Muscles

Elementary motor affections of spastic or paralytic type, limited to a circumscribed region such as the

foot, hand, shoulder, trunk, neck, or a group of muscles, are frequently observed in the neuropathology of the war.

ACRO-PARALYSES AND ACRO-CONTRACTURES (Paralyses and contractures of the extremities of the limbs).

This term describes paralyses and contractures which are limited to the hand or foot. The war has furnished numerous and varied examples.

The exciting cause may be anything from the slightest skin abrasion or contusion to a genuine wound; in other and rarer cases they apparently arise spontaneously without any real cause being found, sometimes preceded by slight joint pains or vague discomfort.

(1) **Paralysis of the hand or foot**, complete and absolute, is rare, paresis is common.

The *hand* is usually limp and flaccid, as in radial paralysis, forming a right angle with the forearm in the extended position. Flexion of the fingers cannot be performed and the thumb alone retains a few limited voluntary movements. When the patient is asked to make some movement, the latter is frequently replaced by a series of wide oscillations —a more or less rapid tremor; this is of considerable interest, as it is absolutely pathognomonic. It is evidence of the contraction of the antagonistic muscles for the attempted movement. A muscle which is incapable of any voluntary contraction may be seen to stand out under the skin, struggling with sudden, brusque efforts against the action of the synergic muscles, which themselves respond not by a regular contraction but by a series of distinctly jerky contractions.

If sharp, passive movements of the forearm are made, the hand follows inertly like that of a dummy figure. Usually the tone of the paralysed muscles remains normal; but in some cases of long standing

we have observed a hypotonicity of the muscles, not so great, however, as that of muscles really paralysed. The hand is often cold, clammy and cyanosed. The skin of the hand and often that of the forearm also is usually found to be analgesic and hypæsthetic to touch.

In the *foot* the paralysis always appears to take the same form, the foot falling into the equino-varus position similar to the club-foot following paralysis of the external popliteal nerve, with "steppage" when walking.

When such a club-foot with equino-varus deformity is carefully examined, it is seen that the point of the foot is dropped and that the sole looks slightly inwards. The patient is incapable not only of making any movement of flexion of the foot, but also any movement of extension. All passive movements are easily performed, especially in old cases where there is slight muscular hypotonicity.

Just as in hysterical hemiplegia and paraplegia, we may find muscular wasting, vaso-motor disturbances and alterations in the electrical reactions (page 720), but they are observed only in cases of profound and long-standing paralysis.

(2) **Contractures of the Hand,** like those of the foot, merit attention on account of their frequency and the importance of their recognition and differential diagnosis. Babinski and Froment, in a series of careful observations, have attempted to separate from the general group of psychoneurotic phenomena a class of so-called *reflex* paralyses and contractures, following either superficial or deep wounds of the affected limbs.

We have to realize that these acro-contractures exhibit such polymorphic forms that it is impossible to describe them completely; each patient, as it were, creates his contracture in his own way, according to his individual personality. There are certain stereotyped forms, however, which are of very frequent occurrence. The *main d'accoucheur* (Meige),

which is the most frequent type of contracture of the hand, had already been fully described by Charcot. The fingers are absolutely rigid, closely approximated, in extension (but with slight flexion of the first phalanx), the thumb extended and adducted with its palmar surface touching that of the index-finger; it is usually impossible to obtain any passive movement. This contracture persists during sleep. If it lasts many months, as often occurs, the fingers waste, the skin becomes loose, cold and clammy. In other cases the four fingers take up the same position, but the thumb is not adducted and the palm is hollowed; this is described as the gutter-like hand (*main en gouttière*).

Neurotic contracture may reproduce, more or less closely, deformities secondary to organic lesions: a *griffe cubitale* affecting the last two fingers, a *griffe totale*, etc. Lastly, in certain rarer cases, the four fingers are immobilized in absolutely complete extension, no passive flexion or any lateral movement being possible. It is a curious fact that the thumb retains complete mobility.

In all these examples the electrical excitability of the nerves and muscles remains practically unaltered, however long the affection has lasted; the sensory disturbance consists of analgesia or anæsthesia.

In **the foot** contracture takes the form of talipes equino-varus, but it is very different from paralytic club-foot. The foot is immobile, fixed and extended, with the point of the foot strongly depressed; the medial border showing an internal curve as if twisted around the axis of the leg; the sole looks inwards and is hollowed by furrows resulting from the deformity. The tendon of the Tibialis Anticus stands out clearly under the skin; the internal malleolus almost disappears, and the external is extremely prominent. The head of the astragalus is also easily recognized.

As a rule no passive movement can be obtained,

FIG. 1.
Hysterical musculo-spiral paralysis.

FIG. 2.—Hysterical contracture of index finger and thumb.

FIG. 3.—Hysterical contracture of the left foot (five months).

FIG. 4.—The same patient two days later, cured.

PLATE II.

Fig. 1. Hysterical Musculo-spiral Paralysis (left). Contracture of the Right Lower Extremity of a Month's Duration. M. H. M. Villejuif Hospital, 5th February 1915.

Wounded on the 2nd January 1915. Scar of a bayonet-wound on the anterior aspect of the right thigh. Scar of a lance-wound on the dorsal aspect of the right foot. Bullet-wound on the palm of the left hand. Left hand dropped and flexed on the forearm, fingers extended. Sensation: glove anæsthesia and analgesia, reaching to the bend of the elbow.

The right lower extremity exhibits paresis and contracture of the thigh and leg. Reflexes normal. No disturbance of sensation.

Discharged cured in May 1915.

Fig. 2. Hysterical Contracture of the Right Index Finger and Thumb. P. E., —— infantry regiment. Villejuif Hospital, 15th January 1915.

Wounded on the 24th August 1914. Shrapnel wound on the external aspect of the right arm. No articular lesion. Contracture of index-finger and thumb in permanent flexion for several months.

The contracture was cured after a single sitting by psycho-electrical treatment.

Figs. 3 and 4. Equino-Varus Contracture of the Left Foot of Five Months' Duration. S. A., aged 21, —— bataillon chass. à pied. Neuro-psychological Centre of D.A.L., 30th August 1916.

Wounded on 16th March 1916 during practice-attacks, at the level of the left internal malleolus. Swelling and adenitis in the inguinal region; a month in hospital. Before abscess-formation, the foot began to show internal rotation: when the abscess healed, the contracture was definitely established.

On admission, irreducible contracture, patellar and Achillis reflexes were brisk on left side; slight wasting of the calf muscles. No appreciable vaso-motor symptoms. Temperature of left foot and leg somewhat raised.

Cure of the contraction after one sitting of psycho-electrical treatment. Pain and swelling lasted during the night, due to fatigue. Left cured on 12th October 1916.

the tibio-tarsal and mid-tarsal joints being immobile; on palpation the antero-external muscles of the leg are quite rigid owing to the excessive contracture. When the patient is told to make some voluntary movement, he is seen to expend a good deal of energy in vain efforts without any degree of success. It is noticed, too, that not only are the muscles of the leg contracted but also those of the thigh. Psycho-neuropathic disturbances of sensation may or may not be present. It is often useless to attempt to test the Achilles jerk on account of the contracture. The knee-jerk is as brisk as on the unaffected side.

When the contracture is of long standing it may be accompanied by skin changes—coldness, looseness of the skin, sweating, cyanosis. These symptoms seem to be due partly to the effect of the contracture on the circulation and partly to the prolonged immobilization. H. Meige, Mme. Benisty, and Mlle. Lévy have shown that the latter may produce a change of temperature of several degrees in a normal individual.

With regard to the circulatory disorders, we have witnessed their complete disappearance after the cessation of the contracture. We shall not refer again to modifications in electrical excitability (slow contraction, tetanic spasm on faradic stimulation), minor degrees of which we have seen in conjunction with lowered temperature.

The **Prognosis** of the psycho-neuropathic acro-contractures is not so favourable as that of the paralyses. They require early and energetic treatment. Otherwise they result in vicious postures with true organic lesions, on account of the relaxation of ligaments, deformities of the articular structures and pains caused by the stretching of nerves and tendons.

Contractures of a Single Muscle or Group of Muscles

We shall mention quite briefly the uncommon but not exceptional cases in which psycho-neuropathic contracture is limited to a single muscle, a muscular group, or even sometimes to a portion only of the muscle. To this category belong the following: Contracture of the *muscles attached to the pelvis and great trochanter*, causing an appearance similar to that of hip-disease; contracture of the *supinator longus*; *contracture of the superior portion of the trapezius* (acromio-clavicular); *contracture of the masseters* (hysterical trismus).

Contracture of the Sterno-Mastoid and Trapezius.—This contracture produces a neuropathic torticollis of the classical type; the head is strongly flexed and more or less markedly rotated towards the unaffected side. This variety of contracture is especially seen as the result of contusions or wounds in the cervical region.

Contracture of the Muscles of the Neck, superficial and deep (psuedo-tetanus, psuedo-meningitis), where the contracture affects symmetrically the trapezius and sterno-mastoid muscles and the deep muscles of the neck. The deformity somewhat resembles that seen in acute meningitis or tetanus, especially as trismus often accompanies it.

The head is immobile and stiff, thrown backwards with the eyes looking upwards and the larynx somewhat prominent. No movement of the head is possible, and attempts at passive movement cause such intense pain that they have to be given up.

Similar pain and contracture may be found in cervical cases of Pott's Disease, in meningeal affections, or in localized tetanus. A careful examination avoids any mistake.

The history shows that the contracture has occurred suddenly, following, it may be, temporary burial in débris in a trench, a contusion or slight wound

PLATE III.

Figs. 1 and 2. Post-Operative Neuropathic "Griffe Cubitale." Amputation of the Second Finger. D. H., —— bataillon chasseurs. Villejuif Hospital, 9th January 1915.

Wounded on 15th September 1914. Compound fracture of the right middle finger at the level of the first phalanx. Amputation of the finger. About 16th November 1914, the right ring and little fingers began to flex; this flexion became more and more pronounced until the palmar surface of the finger-tips eventually became permanently pressed against the palm. No organic lesion of the ulnar nerve.

Fig. 1. Attitude of the hand on admission. Fig. 2. Twenty days later.

Fig. 3. Neuropathic Paralysis of the Hand. H., aged 22. Neur. Centre of Section VIII.

Complete paralysis of the hand following a superficial gunshot wound on the external aspect of the carpus. Glove anæsthesia. No modification of the electrical reactions.

Fig. 4. Neuropathic Contracture of the Hand. C., aged 28. Neur. Centre of Section VIII.

This contracture followed a very slight gunshot wound of the forearm. Electrical reactions normal.

Fig. 5. Club-foot due to Neuropathic Contracture of the Tibialis Anticus and Superficial Calf Muscles following Exposure to Cold. L., aged 25. Neur. Centre of Section VIII.

The patient walked on the external border of the foot. Note the prominence of the tibialis anticus.

FIG. 1.—Hysterical *griffe cubitale*, before treatment.

FIG. 2.
After treatment.

FIGS. 3 and 4.—Two types of main d'accoucheur.

FIG. 5.—Hysterical club-foot, caused by exposure to cold.

FIG. 6.—Post-traumatic hysterical contracture.

of the cervical region. Pain is not produced by pressure on the spinous processes or by percussing the skull, but rather by palpation of the contracted muscles. The complete integrity of the cervical column will definitely exclude Pott's Disease.

With regard to meningitis, in face of the absence of the ordinary series of meningeal symptoms, it is difficult to see how mistakes could be made.

There is greater difficulty in differentiating from abortive or limited tetanus. In one case recorded by Claude and Lhermitte, there was absolute rigidity of the muscles of the back of the neck and the patient, not having received any wound, was put down as a neuropathic subject and as such was sent to the Neurological Centre at Bourges. But in contradistinction to cases of psychoneurotic contracture, his bone and tendon reflexes were profoundly altered. Percussion of the zygoma, occipital bone and clavicle produced a lively contracture of the already contracted muscles. Further, though there was no apparent spasmodic tendency in the lower extremities, ankle clonus, patellar clonus, and definite increase of all the bone and tendon reflexes were found to be present. We may add that in chronic, abortive, or limited cases of tetanus there is hyperexcitability of the nerves and muscles to faradic and galvanic currents (Claude and Lhermitte).

Contractures of the Trunk

These are extremely common and produce the most varied types of spinal deformity: scoliosis, kyphosis, and lordosis. Of all these varieties the commonest is undoubtedly that in which the trunk is curved forwards. It is quite a new type, of which we have seen numerous examples during the war and merits special attention.

Spinal Curvature, Traumatic Kyphosis, Pseudo-Spondylitis, Camptocormia ("Bent Back").—These are the commonest terms used to describe curvature

of the trunk with an anterior concavity. Souques and Mme. Rosanoff-Saloff have suggested that the term kyphosis should not be used, as this incurving of the trunk is not accompanied by any abnormal curvature of the vertebral column, and they have suggested a new name for the condition: *camptocormia* (κάμπτω = I bend, κορμος = the body). The soldiers themselves have given it the term "le cintrage" (arching).

As a result of the concussion of a bursting shell, the soldier is violently thrown to the ground or against the parapet of a trench. After a complete loss of consciousness or only transient stunning, a severe pain becomes localized in the lumbar or sacral region, complete extension of the body being painful. From this time the abnormal posture is established. However long it persists, its characteristics remain the same.

Looked at from the front, the patient holds the trunk almost horizontal, the head generally in a stiff position, the anterior muscles of the neck and the thyroid cartilage projecting; the expression is fixed, the eyes open wide and looking upwards, the lower limbs extended or sometimes semi-flexed. The normal folds caused by flexion of the abdominal wall are exaggerated, and at the level of the groin, the epigastrium and the pubis, there are deep, transverse furrows. Seen from behind the patient presents a series of morphological changes, which consist essentially of the disappearance or flattening of the median lumbar fold, prominence of the spinal muscles (the sacrolumbar, longissimus dorsi group), disappearance of the lateral fossæ over the sacrum, and prominence of the postero-superior iliac spines, lengthening and flattening of the lumbar region. As regards the vertebral column, there is increased convexity in the dorsal region and the muscles are less apparent. The buttocks are flattened and widened transversely. The cervical part of the column is curved backwards and appears much shortened and without any promin-

ence of the seventh spinous process; the nape of the neck is marked by deep transverse furrows.

In uncomplicated forms walking is fairly easy without any limp. Sometimes there is an associated claudication and resemblance to hip-disease (*cf.* p. 784). During progression the trunk and head remain in this stiff, immobile position, which is striking enough in a state of rest.

If the patient is told to stand erect he may be seen to exhaust himself with vain efforts, the face becomes flushed and all the muscles of his body contract, but in spite of all these obvious efforts the bending remains unaltered. If the buttocks are supported by a firm object and attempts are made at passive correction of the trunk, a relative reduction of the curvature is usually obtained, but the correction is never complete. During this manipulation there is evidence of active resistance on the part of the patient; that this actually takes place is shown by the fact that the vertebral and abdominal muscles become still more contracted, as may be readily proved by palpation. If this attempt at correction is made at all forcibly, the patient complains of lumbar and sacral pain, especially the latter, and becomes extremely uneasy; his respiration is embarrassed and quickened, he becomes dyspnœic, the face is terror-stricken, and the pulse becomes rapid; the test has to be given up. When again left to himself, the patient immediately assumes his former attitude and all the phenomena of fear and emotion vanish in a few seconds.

Attempts at correction in the recumbent position give very variable results; in many cases correction is effected with much greater ease than in the upright position and is sometimes complete; but to obtain reduction of the deformity, tactful management is especially needed.

Tests of sensation, of tendon, bone and skin reflexes, show that there is no sign of any organic lesion of the nervous system.

PLATE IV.

Fig. 1. Camptocormia,[1] Antero-Lateral Curvature. B. E., —— infantry regiment. Villejuif Hospital, February 1915.

Wounded on 3rd September 1914. Thrown into the air by a bursting shell : loss of consciousness, followed by severe pain in the back. Trunk bent forwards and to the right since this date. No wound.

In February 1915, application of a plaster jacket, which incompletely corrected the deformity in three weeks (M. Souques). Trunk now only inclined to right, not bent anteriorly.

Second application for three weeks, after which there was complete cure.

Left cured and sent to the school for re-education (Grand-Palais).

Fig. 2. Camptocormia, Anterior Curvature. C. L., —— infantry regiment. Val-de-Grâce and Villejuif Hospital, March 1915.

Buried in débris by a shell on 25th August 1914. No wound or lesion of bone. Large ecchymosis in the lumbar region. Violent pain in the lumbar part of the column. Trunk flexed symmetrically, impossible to correct. On a bed or floor, could be completely reduced.

Plaster jacket three weeks in March 1916 (M. Souques) ; complete correction, maintained after removing the jacket.

Discharged cured.

[1] *Cf.* text, p. 737

PLATE IV.

Can the same be said of the musculature and of the vertebral column ? This is the question that all observers of these curvatures have asked themselves. As regards the muscles, all that is observed is a state of contracture or contraction of the abdominal and sacro-lumbar muscles ; palpation gives evidence of the hardness of the muscles, and attempts at correction with the patient in the standing position are followed by a paroxysm of contracture, but the most minute investigations do not reveal any muscular lesion whatever. It must be remembered that deep pressure on the muscles in the inferior lumbo-sacral region causes little or no pain. As regards the vertebral column, although in certain instances it cannot be examined clinically on account of its complete immobility, in the the majority of cases the passive reduction of the curvature is sufficient to demonstrate the obvious integrity of the vertebral articulations : in making movements of flexion or extension no creaking or crepitation can be heard. Skiagrams also prove in all these cases that the spinal column is absolutely intact.

What is the nature of these curvatures ? Certain authorities (Sicard), arguing from the occurrence of pain and discomfort and from the results of examination of the cerebro-spinal fluid, believe the forward bending of the trunk to be due to anatomical changes in the vertebral column itself (spondylitis), in the ligaments, or the muscles gaining insertion into it (syndesmitis, psoïtis). Sicard thus explains the phenomenon as an antalgic curvature, at any rate in the majority of cases.

Our study of numerous cases of camptocormia over a period of more than two years leads us to differ from this explanation. Indeed, apart from a few rare exceptions, we have found no increase in albumin in the cerebro-spinal fluid. It is doubtful, too, if the discovery of increased albumin in these cases is as important as we have been led to believe. One of our curvature cases with high albumin

content recovered with much greater rapidity than certain others without this phenomenon. May we not consider that in these cases, an increased percentage of albumin is due, not to an organic nature of the affection, but rather to the spinal curvature itself, with the resulting hindrance to the venous and lymphatic circulation? Lastly, the rapid cure which follows energetic treatment, even in long-standing cases, clearly shows the psychoneuropathic basis of the disorder.

Although at the onset it is pain which has almost always been the primary cause of the vicious posture as observed in hospitals near the front (*cf.* p.788), later on this is not the essential element in the curvature; it appears as an associated symptom: the patient becomes deformed not because he is in pain, but he suffers for the very reason of the extremely abnormal position of the trunk which causes pronounced stretching of the vertebral ligaments. This is shown by the fact that as soon as the correction is ultimately obtained, often in a few moments, these patients feel relieved and admit the mitigation and then the disappearance of the lumbosacral pain of which they previously complained. This applies in general to all psychoneurotic contractures.

It should be noticed, on the other hand, that soldiers with evident lesions of the vertebral column caused by gun-shot, shell or contusion do not exhibit curvature though this might seem likely at first sight. The same applies to cases of spinal osteitis due to Pott's disease or vertebral cancer, where in spite of the intensity of the pain (pseudo-neuralgia), a posture of the type of these curvatures of war is never seen.

Diagnosis.—All cases of curvature following concussion or vertebral contusion are not purely neuropathic. In some of them there may be evidence of a latent organic lesion, the result of trauma, or even

the production of some definite osseous or articular lesion.

Let us briefly recall the possible sources of error: *Pott's Disease* may be latent and only come to light on the occurrence of some spinal trauma; diagnosis is often a delicate matter and demands minute investigation; but in such cases it is usually a question of the vertebral column being fixed in an upright position and not with curvature. The discovery of definite painful points over the spine, examination of the cerebro-spinal fluid and later on the signs of compression of the spinal cord and nerve roots will prevent errors of diagnosis.

There is greater difficulty in the differential diagnosis of hysterical curvature from *traumatic spondylitis* or *Kummel's disease*: forward curvature of the trunk following trauma. In this case the curvature does not follow until several months after the spinal contusion, and is, moreover, preceded by a period of pseudo-neuralgia in the nerve roots; the dorso-lumbar kyphosis is pathological.

Vertebral Ankylosis (Bechterew) and the *Rhizomyelic Spondylosis* described by Pierre Marie may also result from trauma. But in these conditions it is not the trunk which is flexed, but the vertebral column which is incurved and immobile; the deformity develops slowly and the disease is progressive; in Pierre Marie's disease the shoulder-joints or hip-joints are affected.

Bruising of the Intervertebral Discs (Kocher) causes pronounced difficulty in maintaining the upright position and in walking, with local and radiating pain; it is also characterized by localized œdema over the spines of the affected vertebræ and cure takes place by synostosis.

Myogenic Ankylosis of the Vertebral Column (Schuster) is distinguished by disappearance of the normal spinal curves and is accompanied by paresis, increased tendon reflexes and muscular wasting, all of which are absent in camptocormia.

PLATE V.

Figs. 1 and 2. Vicious Posture and Limping of the Hip-Disease Type (right), of Four Months' Duration, following a Superficial Wound in the Right Flank. M. L., aged 36, —— infantry regiment. Neurological Centre of ——th Army, 16th November 1916.

Wounded on the 9th June 1916 by a machine-gun bullet in the superficial tissues of the right flank. The patient was said to have suffered and limped since July, from the time when he first left his bed. Several nervous crises since being wounded; forty-five days' convalescence. Request for extension of sick-leave refused; stayed in a neighbouring dépôt for affections of the gait, whence he was sent back for treatment.

Pronounced limping of the type seen in hip-disease, trunk inclined slightly forwards and to the right, fold of the right groin strongly marked. Right foot only touches the ground anteriorly; the knee is held slightly flexed. Respiration panting on walking. Tendon and skin reflexes brisk. No disturbance of objective sensations, but sharp pain referred to the situation of the scar.

Isolated for two days, then treated by psycho-electrical methods; violent defensive reactions; at the end of this single séance of treatment walking was carried out normally. Retained for six weeks to make certain that cure was permanent. Left 30th December 1916.

Fig 1. Appearance of patient on admission. Fig. 2. Three days later, cured.

Figs. 3 and 4. Long-standing Sciatica of Three Months' Duration. D. M., aged 33. Neurolog. Centre of D.A.L., 13th August 1916.

Very pronounced limping, trunk considerably inclined forwards and to the right, left knee not flexed when walking, all the toes dorsally flexed and not touching the ground. Lasègue's sign. No alteration in tendon reflexes.

No improvement after epidural injections and the usual methods of treatment. After one sitting of energetic faradic stimulation, the gait became about normal and the vicious posture was corrected. Absolute cure in a few days.

FIGS. 1 and 2.—Vicious posture and limping of the hip-disease type (right side) of four months' duration. Left: appearance of patient on admission. Right: after cure.

FIGS. 3 and 4.—Long-standing sciatica of three months' duration (right side). Left: patient before treatment. Right: after treatment.

Lordosis from Contracture of the Dorsal Muscles.—Backward bending of the trunk is as rare as the forward bending is common. The reason must be sought for in the general law of contractures, according to which deformity is usually produced in conformity with the most powerful of the muscles acting on the part; possibly also in the fact that to avoid injury from falling objects the instinctive protective movement is one of bending forward; lastly, in the fact that hysteria usually copies such attitudes as are the easiest to reproduce or to maintain voluntarily.

The patient's trunk is bent backwards, the head is flexed and the expression is fixed. Walking is performed with ease, but during progression the trunk is immobile and fixed. Correction, forward flexion of the trunk, is almost impossible in the standing posture, but is quite easily performed in the recumbent position. This contracture, also, is accompanied by pain in the loins and back.

Scoliosis from Contracture of the Abdominal and Sacro-lumbar Muscles.—Lateral flexion of the spinal column by contracture, although rarer than lordosis and kyphosis of the trunk of traumatic origin, and resulting from temporary burial for example, is frequently observed as a habit-posture following long-standing sciatica. We have seen examples of this which have been completely corrected by re-education.

In the pure neuropathic cases the trunk is considerably bent over either to the right or left, the cutaneous folds of the flank being strongly marked and the lower ribs coming into contact with the iliac crest. Examining the patient from behind, the scapula and upper extremity are found to be depressed, the normal curve of the dorsal column and the compensatory cervical curve unaltered. The dorso- and sacro-lumbar muscles in the concavity of the vertebral column are prominent and hard to the

touch, obviously contracted. The serratus magnus and the oblique muscles of the anterior abdominal wall appear in an evident condition of hypertonicity on palpation.

We have found that correction of this deformity is more readily obtained than that of lordosis and kyphosis, and permanent cure also seems easier to effect.

The contractures we have outlined in the preceding pages will be seen to present a whole series of common characteristics, which we believe justify us in including them in the same nosological category and ascribing to them the same pathogenesis.

The course of these conditions forms an additional bond of union. It is true that their cure is frequently but not always more difficult to obtain than that of the paralyses; however, these contractures disappear under the influence of counter-suggestion, persuasion and other means, but with the proviso that physical re-education takes a prominent place amongst the therapeutic measures employed. The reason is that here, as in the other motor conditions, a mental element is added to the muscular disorder, which is the essential factor from our point of view.

Differential Diagnosis and Nature of Psycho-Neuropathic Contractures in General

We shall not dwell on the differential diagnosis of localized contractures and those secondary to organic affections of the central nervous system.

In an acro-contracture, or one still more limited, the only problem which presents itself is to decide on the neuritic, muscular, reflex or psycho-neuropathic nature of the deformity.

It is known (Volkmann) that the application of too tight a bandage may be followed by a retraction of the muscles of the hand or foot after considerable pain, which at first sight simulates functional con-

tracture. But in this instance the deformity of the hand or foot is not due to contracture, but to the muscular retraction brought on by anatomical changes of the muscles, whose supply of fluid has been interfered with for too long a period. The retracted muscles are hard to the touch and painful. In case of doubt, spinal anæsthesia (for the lower limb) or a general anæsthetic will settle the diagnosis by demonstrating either the complete disappearance of the contracture or the absolute irreducibility of the retraction.

André Thomas and G. Guillain have shown that in certain peripheral nerve lesions, not paralyses but contractures may be observed, phenomena which may bring to mind fibro-tendinous retraction or psycho-neuropathic contracture.

We have seen a few cases of this kind, but they are extremely infrequent. These contractures especially affect the hand and forearm, when the question arises of a lesion of the radial, the median, and, exceptionally, of the ulnar nerve. The distinguishing point that at once characterizes this form of hypertonicity is that it does not affect muscular groups at random as in neuropathic contracture, but that it is limited to a group of muscles having a common nerve-supply; also that its appearance is preceded by a phase of paralysis.

We will again mention that psycho-neuropathic contracture is usually not limited to a group of muscles supplied by a single nerve and that it occurs either immediately or very soon after the initial trauma.

When the hypertonicity is secondary to a nerve lesion we always find more or less definite changes in the electrical reactions of the muscles and objective disturbances of sensation: hyperæsthesia, tingling on touch, paræsthesia. As the pathogenesis of these contractures secondary to lesions of nerve-trunks is still obscure, we think they should be considered as the expression of an irritation of the motor nerve and not as a reflex contracture.

The differential diagnosis of *psycho-neuropathic contractures* from the so-called *reflex contractures* that Babinski and Froment have specially studied, is a much more delicate matter. Without entering into a discussion which would be out of place in this connection, we must remark that the diagnosis of contracture or paralysis of reflex type ought not to be carelessly applied on the sole discovery of a condition of contracture or paralysis secondary to a lesion of a limb or of the body. Such a contracture or paralysis necessarily presents special clinical characters and may be in keeping with an evident lesion of part of the affected limb. It is quite common, for example, to see contractures of a reflex order accompanying articular, osseous and muscular lesions; and, owing to the researches of Hunter, Charcot, and Dubreuil, they constitute an important factor in diagnosis and are often a valuable guide in seeking for some lesion which is not apparent at first sight.

The same considerations hold good for contractures which result from the irritation produced by the presence of a foreign body adjacent to branches of nerves.

Babinski and Froment have made a careful analysis of these "reflex" paralyses and contractures, better styled "physiopathic"; and have ascribed to them a series of features which allow of their complete differentiation from similar manifestations of a psycho-neuropathic order.

These contractures or paretic conditions have this common characteristic: they are sharply localized in a segment of a limb or in certain muscular groups —usually of the extremities. Though the tendon and bone reflexes may not be modified to any extent, there is generally a condition of mechanical hypersusceptibility of the muscles of the affected limb, with a corresponding hyperexcitability to faradic and galvanic currents with rapid fusion of the contractions (premature tetanus) or sometimes slightly decreased excitability. Occasionally the nerves

themselves also exhibit this hyperexcitability to electrical and mechanical stimuli.

These modifications of excitability are partly the consequence of the lowered temperature of the wounded limb (Babinski and Froment); they improve on the application of heat and are exaggerated by cooling. Like all the contractures, they disappear under anæsthesia; but it is often only under deep narcosis that they disappear. Chloroform anæsthesia, on the other hand, brings to light the hyperexcitability of the medullary centres, causing a selective exaggeration of the tendon and bone reflexes in the affected limb and in it alone. Lastly, important vaso-motor symptoms are of frequent occurrence: cyanosis, lowering of temperature of the extremity by several degrees, diminished amplitude of the arterial pulse measured on a Pachon's sphygmomanometer; also disturbances of the sweat-glands and decalcification of the bones. The latter phenomena, according to Babinski and Froment, reach a degree of severity much greater than the maximum seen in old-standing neuropathic conditions.

With regard to *paralyses of a reflex type*, these are characterized by changes in excitability of the muscles to faradic, galvanic and mechanical stimulation, by decreased muscular tone, and often by muscular wasting. It should be noticed that although it is well known that reflex amyotrophy secondary to arthritis or an osseous lesion is usually accompanied by definite exaggeration of the tendon and osseous reflexes, in the above conditions Babinski and Froment show that there may be no modification of the reflexes.

These paralyses and contractures of a reflex type are, in their nature, very resistant to treatment by psychotherapy.

A condition very similar to Babinski and Froment's reflex contracture is the affection described by Pierre Marie and Foix under the name of *paratonic paresis*

of the motor muscles of the hand; the hand, flexed on the forearm by contraction of the palmaris muscles, presents a deformity analogous to that of the *main d'accoucheur,* or like a swan's neck; the fingers are inflexible and difficult to mobilize.

To sum up, the diagnoses of localized paralyses and contractures presents, in the great majority of cases, no real difficulty. We cannot, however, even in cases which seem most definitely psycho-neuropathic, insist too strongly on the necessity of making a methodical examination both of the reflexes and the electrical reactions. Combinations of hysterical or neuropathic and organic causes are so frequent that only the careful observer can differentiate that which arises from a muscular, nervous, articular, osseous, or vascular lesion from that which is attributable to a neurosis.

With regard to *paralyses or contractures of reflex type*, these seem to be rare, and the clinical signs which Babinski and Froment have attributed to them are not sufficiently characteristic. Such symptoms, indeed, are frequently found in cases of paralysis or contracture of fairly long standing and are secondary to lowered temperature, venous stasis, and immobilization. Besides, the term "physiopathic" paralyses and contractures, more recently employed by these authorities, is much more exact and avoids controversy.

As Claude has shown, many paralyses or contractures at their onset may remain for several hours of a reflex nature, but the fixation of the motor affection is effected by the psycho-neuropathic state. The best proof we can adduce of our argument is that *all* the signs described by Babinski and Froment as characteristic of reflex disorders may be found in paralyses and contractures *without any bodily wound,* and that they *disappear* under treatment by psychotherapy (Claude and Lhermitte).

Chapter II

PSYCHO-NEUROPATHIC DISTURBANCES OF THE GAIT

The psycho-neuropathic disturbances, which may affect the action of walking, have appeared in diverse and varied forms during the course of the war. We have already studied several types in the sections on hemiplegia, crural monoplegia and paraplegia.

The function of walking, when analysed, appears extremely complex. For its correct performance it is necessary that not only must the motor and sensory functions be intact, but also the cerebellar co-ordinative mechanism must not be disturbed, nor the visual or auditory apparatus, by means of which we determine our position in space. The very complexity of the systems of equilibration and co-ordination accounts, in part at any rate, for the readiness with which their functions are disturbed in the psychoneuroses. Further, if the function of walking, after having been a voluntary and conscious act, becomes by its very repetition an automatic performance, it must be remembered that this automatism is on the same level as consciousness in that it is highly susceptible to the psychological changes, the sentiments, the emotions, and the suggestions which are the basis of the psychoneuroses.[1]

[1] In a recent monograph, Laignel-Lavastine and Courbon (Paris Médical, 2nd Sept. 1916) divide the functional disorders of the gait into three groups: the first is characterized by dynamogenic phenomena, the second by inhibitory disturbances, and the third by a mixture of the two. Attractive though it may appear, this scheme seems difficult to apply to disorders of the gait, because of the mingling in each separate case of dynamogenic and inhibitory

The study of functional disorders of the gait shows that one class of these has as a psychological substratum an active emotional condition, the other a condition of suggestibility of which the clearest expression is the fixation of the disturbance of a bodily function.

We shall use the term *basophobic disorders* to describe the former, *abasic or dysbasic disorders* the latter.

§ 1. Astasia-Abasia ; Dysbasia

Astasia-Abasia.—This syndrome, described by Jaccoud, Blocq, Charcot and others, is one of the commonest seen during the war ; although rare in the complete form, it is common in diminished or abnormal forms.

Usually following the shock of explosion of a projectile of large calibre, its onset is rapid ; and as emotional and concussion phenomena are not in evidence it is easily recognized.

It commonly occurs in a man who has been thrown to the ground more or less violently and who has rolled into a trench or hollow. He has sometimes been able to get back to the first-aid post either by laborious walking or by crawling along the ground. By the time he gets to the ambulance he is quite unable to walk.

When he is examined lying down he exhibits no elementary motor disorders : all movements, muscular power and tendon, osseous and cutaneous reflexes are normal.

If placed in the upright position he either collapses on his flaccid lower limbs or the latter are shaken by a rapid tremor which ends in an indefinite treading movement. When told to walk, his limbs are incapable of action, and in spite of an obvious effort

elements, and because of the uncertainty we always experience as to the exact nature of the psycho-neuropathic phenomena which disturb the automatic performance of the act of walking.

his legs are quite unable to execute a voluntary movement. He is unable to walk even with the aid of two sticks; the limbs are dragged along inertly, as in true paraplegia. In other cases there is an extremely pronounced contracture of the lower limbs which seem to resist the execution of any movement of walking.

Dysbasia of Choreiform or Saltatory Type.—The gait is inco-ordinate, the lower limbs are put forward asymmetrically, as in the next type, but in this variety walking is still more inaccurate; not only are the alternate movements of the lower limbs irregular and asymmetrical, but also spontaneous, "choreiform" movements not having any connection with the object in view are superimposed on the automatic movement of walking. When standing erect, the muscular disorder may be quite obvious, but is always exaggerated on walking (dysbasia with tremor). If the upper extremities are not involved cases may be observed in which, during standing or walking, the arms are shaken by brusque and jerky movements of flexion and extension, and the hands are never at rest.

Dysbasia of Pseudo-Tabetic Type.—Here again all movements of the lower limbs are carried out normally in the recumbent position. When walking, however, the patient progresses throwing forward the lower limbs; he kicks his heels, swings the limbs and widens his base. At first sight it looks like true ataxia. It should be noticed that forward progression is effected without the individual experiencing the least discomfort or the least fear. When placed upright, irregular, wide oscillations, both antero-posteriorly and laterally, are observed, not exaggerated by closing or covering the eyes. The malprogression is not exaggerated in the dark.

Dysbasia of Pseudo-polyneuritic Type.—Just as the last condition roughly resembles that of ataxia,

PLATE VI.

Figs. 1 and 2. Astasia-Abasia with Tremor. V. A., —— infantry regiment. Villejuif Hospital, 20th March 1916.

Wounded on 23rd September 1914. Gunshot wound in the middle of the left thigh, anteriorly. Unable to walk from the moment he was wounded ; then he was gradually able to hold himself erect and to walk. Returned to the front in January 1915.

Again wounded on 6th January 1915 ; this time a slight lesion in the nape of the neck. Sent to hospital and an operation performed. From this time unable to walk or stand upright. Reflexes normal. Normal motility when the patient was lying down, but movements were performed slowly. When placed in the upright position the patient was seized with tremors and could not maintain the vertical posture nor make a single step forward. Using a pair of crutches, he dragged both legs after him.

After electrical treatment (faradic current, wire brush) he was able to stand upright and walk. Cure after one séance of treatment.

Figs. 3 and 4. Astasia-Abasia due to Shell-Shock. P. G., —— infantry regiment. Villejuif Hospital, 8th July 1915.

Wounded in September 1914. Superficial wound of the thoracic wall under the right nipple. Thrown into a very deep shell-crater. He reached the field-ambulance without help but could only walk with short steps. From this time, spasmodic, tremulous and hesitating gait. Using two sticks, could only walk laboriously and with a tremor. At each step forward, swung the body forwards, only taking short and very irregular steps, and gave the impression of a man drawing a vehicle and at each step making a great effort to advance.

One séance of electrical treatment (faradic current, wire brush) cured the patient.

Left : Appearance and gait on admission. Right : Cured.

Astasia-abasia.

FIG. 1.—Mode of progression on admission.

FIG. 2. Cured.

Astasia-abasia with tremor.

FIG. 3.—Spastic and tremulous gait on admission.

FIG. 4. Gait after cure.

this type has certain features in common with the complex condition seen in polyneuritis of the lower extremities on the way to recovery, the disorder having both a paralytic and an ataxic element. This seems also to be the case in certain types of psycho-neuropathic astasia-abasia. The patient can hold himself upright fairly easily, although sometimes he first shifts his ground as if seeking to find his centre of gravity. Forward progression is characteristic: the thigh is unduly flexed, while the leg remains vertical, the foot slightly dropped as in the "steppage" gait. The limb which is advanced comes heavily to the ground. Thus progression takes place with exaggerated "steppage" and abruptness of movements.

Atypical Forms.—In addition to the types we have just described and which seem to be the most often observed, astasia-abasia may take the form of other affections of the gait, a complete description of which is impossible by reason of their extreme diversity. We will only mention the most distinct varieties.

(1) *Tight-rope walker's gait.*—The upright posture is practically correct; even with the feet together the patient is steady enough on his legs, but when he is asked to walk his lower limbs become stiff. He makes obvious efforts. Progression is effected exactly in the manner of a tight-rope walker. With eyes fixed straight in front of him and tense features, the patient advances by small steps, placing the feet alternately one in front of the other. On account of the contracture of the lower limbs, which gives him an unstable equilibrium, progression is difficult and he holds out his arms as if holding a balancing-pole; in spite of this, his equilibrium is imperfect and he is obliged to make three steps to the right or left, after which he again continues his advance.

(2) *Knock-kneed gait.*—A special type of gait observed in one of our own cases. Nothing abnormal

PLATE VII.

Figs. 1 and 2. Limping of the Hip-Disease Type (left side) and forward Curvature of the Trunk, of a Year's Duration. M. P., aged 29, ——th chasseurs. Neuro-psych. Centre of D.A.L., 2nd September 1916.

Wounded by shell in the left flank and buried on 29th July 1915; loss of consciousness, embarrassment of respiration and mutism. Walked bent forwards and limping since 25th August 1915.

Several times in hospital and six months at the depôt. Sent to the front on 20th June 1916 after having been recommended for light auxiliary service at the divisional depôt. Underlying mental deficiency. By one séance of electrical treatment the vicious posture of the trunk was corrected. Limping persisted and necessitated prolonged daily treatment by re-educative methods.

Discharged cured on 20th October 1916, without limp or curvature; slight lumbar pain persisted. Given sick-leave.

Left: Posture of patient on admission. Right: Eight days later, cured.

Figs. 3 and 4. Hysterical Paraplegia, "Tripod" Progression; Rigidity of the Lumbar Vertebræ, lasting Six Months. J. G., aged 45, —— territ. · Neur. Centre of D.A.L., 28th August 1916.

Spontaneous onset as a result of exposure to cold, and diarrhœa followed by constipation; the curvature and affection of the gait developed progressively in the casualty clearing station.

Arrived carried on a stretcher; walking extremely difficult, by means of holding on to a stick with both hands; tremor of both legs, which were in a pseudo-spastic condition.

Psycho-electrical treatment on the following day, resulting in complete cure. Mental deficiency and constitutionally neurotic.

Discharged cured on 20th October 1916, on sick-leave.

Limping of the hip-disease type with curvature of a year's duration.

FIG. 1.—Appearance of patient on admission.

FIG. 2. Cured.

FIGS. 3 and 4.—Hysterical paraplegia, "tripod" progression, with lumbar contracture.

when at rest, standing upright; but when the patient commenced to walk, the lower extremity became flexed and the knee turned inwards at each step, the trunk plunging forwards and inclining to the side of the leg supporting it.

(3) *Walking as if on a sticky surface* (Laignel-Lavastine and Courbon).—The patient advances by sudden steps, the back arched and the hands applied to the hips. He walks as if on a sticky surface.

(4) *Bather's gait* (Laignel-Lavastine and Courbon).—This gait resembles the movements of a man struggling against the tide, advancing into the sea. He throws back his body and raises his legs laboriously.

(5) *Scrubber's gait* (Laignel-Lavastine and Courbon).—The legs are extended; the foot is not raised from the ground, on which it rubs and drags along. He seems to drag along as if with rivets in his ankles.

§ 2. Staso-basophobia

Although this roughly resembles astasia-abasia, and although the functional disorder causing it produces much the same tangible result, *i.e.* impossibility of standing and walking, its intimate mechanism differs widely from that of true astasia-abasia. The latter is a disturbance of the automatic performance of walking, the former is an inhibition of this automatism dependent on an emotional condition. And although astasia-abasia and staso-basophobia both belong to the psychoneuroses, the former come within the category of the hysterias, the latter that of the phobias, hyper-emotions and obsessions.

Further, in spite of their apparent resemblance, how different these two conditions really are! In the recumbent position elementary movements are performed almost normally. When placed upright the patient's facial expression immediately changes and instead of appearing calm, he shows fright, anxiety and terror. He begs to be held up, says he is going to fall and that everything is revolving round him.

He clutches at the sticks which hold him up or clings desperately to the furniture around him. There is no question of making him walk—he can only be made to take a single step with difficulty: if he puts one leg forward he quickly draws it back, certain that he is about to fall.

Sometimes the clinical picture is less dramatic and, strongly supported, the patient succeeds in slowly and timidly moving his lower limbs. The feet are uneasily raised from or slip along the ground; often, in spite of his efforts, the legs flex, the foot does not entirely reach the ground and is a new source of distress. Pale, trembling, and bathed in perspiration, the patient begs to be allowed to rest.

Both in the case of staso-basophobia and astasia-abasia, these patients are always favourably influenced by psychotherapy; methodically and vigorously carried out, psychical re-education, aided more or less by the usual methods of physical training, results in a cure of these psycho-neuropathic affections of the gait.

Diagnosis.—Affections of the gait have many origins, both organic and functional. Thus in any given case the question must be considered as to whether the disorder is provoked by some definite lesion or by some purely functional condition. Simple though this may seem at first sight, it is often far from an easy matter, especially in cases where both elements are concerned.

The organic affections of the nervous system which produce affections of the gait are too numerous and well known to be described here.

Besides, mistakes should not be made given a methodical examination of the sensory, motor, and reflex functions and of the vestibular and cerebellar mechanisms of equilibration. There is much greater difficulty with cases in which psycho-neuropathic disorders are added to organic lesions, and their recognition is a hard problem. According to the

personal tendency of the observer, we have often seen patients exhibiting both elements diagnosed sometimes as organic and sometimes as purely neuropathic. This failure of recognition is, however, important and ought not to be considered lightly; it is of essential and practical importance, since on the solution of this problem depends the decision of the military value of the soldier.

We will only stop to mention functional disorders of the gait resulting from lesions of the vestibular apparatus. The war has shown frequent examples of labyrinthine disturbances following the explosion of large projectiles. Since the vestibular apparatus is the essential organ of equilibration, one can easily understand the frequency of disorders of station and locomotion following cerebral concussion or wounds of the cranium, as Moutier, Lhermitte and Cestan have pointed out. But although the cerebellar syndrome may be more or less clear in the psychoneuropathic phenomena, the vestibular syndrome is very frequently masked by astasia-abasia or stasobasophobia. We have ourselves observed several cases of wounded men in whom there existed without doubt a labyrinthine lesion following concussion; but only testing the vestibular branch of the eighth nerve by the most delicate methods will reveal these slight lesions (galvanism, Bárány's experiment, etc.). In spite of the minor degree of modification of the vestibular function, these patients were incapable of walking and even of maintaining the erect position, without assistance.

Guillain and Barré have quite recently observed the phenomena of astasia-abasia in patients with specific lesions of the labyrinth, which do not differ from those we have described.

§ 3. Habit-limping

Cases which come under this heading are, according to our experience, very numerous; the subject

had been little understood or studied in France before the war. H. Meige, in October 1915, drew attention to these cases at the Paris Neurological Society.

In previous sections, dealing with the elementary motor disorders—neuropathic hemiplegia or paraplegia, we have mentioned the so-called " habit paralyses " (Ehrer).

But in studying disorders of the function of walking we must describe separately " habit-limping." They possess a real and important interest by reason of their frequency, by the fact that they are imperfectly understood or even ignored by the physician and because of the difficulties of diagnosis and of interpretation which they exhibit.

Their causes are those which, at a given moment, have been the origin of functional impotence of a lower limb : wound, fracture with prolonged immobilization and extension apparatus, painful affections of the limbs, arthritis of the hip-joint or knee, rheumatism and more especially sciatica and neuritis.

In short, an organic cause, often very slight, produces an antalgic position, an abnormal attitude, a defective use of a limb, or even contracture of certain groups of muscles, conferring on the limb a vicious posture with an affection of the gait. This abnormal method of walking gradually becomes automatic ; the patient more or less unconsciously is unable to alter it ; it has passed into the sphere of the unconscious and become a " habit." This is the only remnant of the functional disorder, and the individual finds it a matter of great difficulty or even impossibility to overcome it by himself. As Meige puts it, " Habit creates aptitude towards an attitude."

The vicious posture at first behaves as one would expect : if such a patient flexes the leg when walking, it is because the heel is painful or there is discomfort along the course of the sciatic nerve, for example. The abnormality lies in the fact that the posture is

indefinitely prolonged after the organic cause has long disappeared.

The morphological and clinical aspect of these cases is so extremely variable that we cannot give a common description of them. We have had and shall again have occasion to refer to them several times in the course of this book in dealing with motor or sensory disorders of neuropathic type. Moreover, the subject is one little understood.

We will only say for the time being that one cannot attach too much importance to the minute observation of all the motor actions which direct the very complex automatic movements of walking.

In a methodical analysis of the gait and of the causes which disturb it and promote limping, one often succeeds, unknown to the patient, in discovering some features which are illogical and paradoxical, not in accord with the lack of power or the pain of which the patient complains; such signs may give a clue to the diagnosis. Sometimes the limp is characterized by the simple irregularity in the length of the paces taken or in the length of time during which one of the extremities is applied to the ground. Sometimes, and this more often, it is by a physiological disuse of one of the joints, the freedom of movement of which is limited by contractures or a vicious posture: walking on the toes, or flat on the foot (the heel and ball of the great toe both touching the ground at the same time), or with leg and foot turned outwards, etc. A type of limping which belongs to the same category and seems to be particularly common is *claudication with dorsal flexion of the toes*; R. Leroux and one of the present authors have drawn attention to this condition. It occurs in patients who have had somewhat vague, "rheumatoid" pain, sciatica, or frost-bitten feet with so-called neuritic symptoms and who maintain the claudication for many months; the limp is due to the fact that the patient applies the ball of the foot to the ground but lightly and the toes fail to reach

it at all, being maintained in almost constant dorsal hyperflexion. Walking is carried out, as regards the affected foot, as if the patient were walking barefoot over a surface covered with small stones.

Changes may also be observed in which he balances himself by the use of one arm, the other arm often remaining immobile, or with vicious postures of the head or trunk. In short, it is one or several of the complex actions of walking which are affected—a principal action (movements of the lower limbs) or an accessory action (equilibratory movements of the trunk, head, or arms, etc.). All these affections are exaggerated by running.

But apart from the paradoxical movements mentioned above, we have not shown the differentiation of prolonged and old-standing habit-claudication and recent, true claudication of organic origin. The point that characterizes the former is that frequently nothing is found to account for the claudication when the patient is lying in the recumbent position, at rest on a bed ; it is only while standing upright or walking that the vicious posture or motor phenomena make their appearance. Various methods can be used to render evident the " illogical " character of these claudications. One of these is the *marche à genoux* (Meige) in cases of limping by contracture of the muscles of the hip and tilting of the pelvis. Disconcerted by this unusual method of walking, the patient—at any rate at the commencement of the test—" forgets " his vicious posture and his limping.

We have often observed this occurrence in walking with changes of step, in walking backwards, and in walking or running to a certain fixed spot. These tests serve both as methods of examination and useful means of re-education. The automatism recently created to fit in with a vicious gait has not become sufficiently fixed in the realm of the subconscious to become spontaneously adapted to

complex and unusual motor actions, such as the above.

The nature of these disorders is difficult to define precisely, especially as their origin appears to be connected with various causes.

On the basis of cases actually observed, three different types of patients can be described :

(*a*) In some cases the claudication is only the effect of a contracture, pain, or a hysterical paralysis, little evident or not apparent except when walking or in the upright position, but revealing itself on the slightest attempt at progression. To this class belong the cases we have just described. The disorders of the gait are only secondary to a vicious posture of the limb or body ; at the most, they only make for its maintenance or reinforcement and for this reason alone come within the scope of our subject. They are neuropathic patients in whom the vicious posture must be treated and cured ; with its disappearance the disorders of the gait also vanish.

(*b*) In other cases pain or functional paralysis, muscular weakness, stiffness of joints become considerably improved and do not exist, even according to the patients' own statements, except when walking. In spite of this, the claudication persists in a well-marked degree. The individual limps because he is in pain, but in reality the reverse is the case : he suffers because he limps. The vicious posture and abnormal gait which have become a habit created by fear of pain are really the principal phenomena, which cause or maintain the pain. This is well shown by the fact that the latter vanishes with the claudication under the influence of psychotherapy and psychomotor re-education. In numerous cases, by these means we have been able to obtain definite cure of limping that has lasted many months.

These are true psychopathic conditions, minor

obsessions on the same level—except for slight shades of distinction—as staso-basophobia. Also it is frequently individuals in a state of mental weakness who persist in maintaining claudication.

(*c*) In a third class of patients we have to recognize a wilful inertia; they make no attempt to modify their disorder, prolonging or even exaggerating or simulating an affection. These, of course, are pure malingerers, amenable to stern treatment. Cases in this category appear to be rare compared with the two preceding groups.

Finally, in settling the diagnosis of habit-claudication, the greatest care must be taken to make a minute examination of all the locomotor or nervous mechanisms which control the function of walking, in order to exclude any organic affection: such are the condition of the reflexes, the trophic condition of the muscles, electrical reactions, condition of the joints, skiagrams, etc. A test of the reflexes under chloroform anæsthesia should also be made on the lines suggested by Babinski and Froment; asymmetrical accentuation, disappearance and especially asymmetrical reappearance of the tendon reflexes, particularly the reflex nearest to the affected joint, clinch the diagnosis in favour of an organic lesion according to the above authorities.

This can only be done by repeated examinations, and it is only when all possibility of organic mischief has been excluded that one is justified in making the diagnosis of habit-limping.

Lastly, it must not be forgotten that these conditions, when prolonged for a certain length of time, may, like neuropathic paralyses and contractures, engender organic changes such as shortening of tendons, limitation of articular movement and arthritis, which will prove more or less intractable.

Chapter III

TREMORS, TICS AND CHOREIFORM MOVEMENTS

Though the nervous disorders studied in this chapter are among the less important of the psychoneuroses of war compared with those already described, yet they are by no means rare.

Since the commencement of the campaign many soldiers exhibiting these affections as a result of shock or injury have been treated in the various neurological departments at the front and in the interior; in many of them the condition has persisted for a long time. The long duration of these disorders has led to the question being raised by Babinski at the Paris Neurological Society as to whether a certain proportion of these tremors should not be included in the category of organic diseases and placed beside the major affections of the nervous system characterized by tremors and based on a central lesion.

In order of frequency, we shall first discuss tremors, then spasmodic tic and choreiform movements.

The conditions under which the symptoms appear are much the same in all; they will therefore be taken together.

Sometimes it is a young soldier arriving at the firing-line for the first time; at other times it is a soldier already hardened to the campaign and who has been under a violent bombardment in some specially hard-fought action.

In some cases these patients will be found to

be particularly nervous, emotional individuals with previous neuropathic traits; but more commonly tremor or tic will appear in soldiers previously quite sound, without any neuropathic or psychical history.

We only mention these questions of etiology in passing, as they are discussed more fully in a later chapter dealing with the general etiology of the psychoneuroses of war (p. 870).

We must signalize one important and unexpected fact: the rarity of immediate, direct contagion. In our own personal observations and in those recorded by other writers, there are records of several soldiers in the same section or company simultaneously affected by tremors, tic, or, choreiform movements; but though these instances exist, they appear to be exceptional.

These patients are, in the immense majority of cases, isolated examples. The disorder appears as a sequel to nervous shock, following a shell explosion. In rarer instances, it has followed some slight wound by a bullet or shrapnel; or it has been of spontaneous origin after fatigue, exposure to cold, or a rigor (a case of our own coming within the last class).

Their **Onset** is somewhat variable. It is generally *early*. A man is in a trench, at a listening-post, in a communication-trench or a rest-billet, and is submitted to the explosion of a projectile at short range, such as an aerial torpedo, a mine, or large-calibre shell. He is thrown into the air, falls to the ground or is deeply buried with some of his comrades and loses consciousness. When he comes to himself at the field ambulance or casualty clearing station, he is seized with a tremor or clonic movements. More rarely, the symptoms appear without previous loss of consciousness, immediately after the explosion, at the moment when the man rises or attempts to do so.

In certain cases the clonic movements are accompanied by other concussion disorders, the commonest of which is deaf-mutism.

FIG. 1.—Contracture of the right knee of eighteen months' duration.

FIG. 2.—The same patient cured after one séance of treatment.

FIG. 3.—Neuropathic scoliosis, following a slight wound in the right flank.

Fig. 4.
Clonic tic of the left shoulder.

Occasionally it is of *late* onset ; the symptoms only appear after a lapse of some hours or days after the trauma (*phase de méditation* of Charcot). It may be that the patient only commences to tremble when he leaves his bed with his injuries healed. In the latter case there is some reminiscence of the previous shock : *retrospective emotion.*

Various Clinical Types.—We shall study these in order :

(*a*) The tremors, including the various choreiform movements.

(*b*) The tics.

§ 1. The Tremors

These may be divided into two classes according to their clinical types :

Atypical tremors, consisting of disorderly, irregular movements not resembling in their behaviour or course any tremors accompanying known maladies.

Typical tremors, which more or less resemble those seen in those nervous diseases which are accompanied by tremor.

The former seem to be entirely conceived by the individual, who appears to determine as he wishes their form and manner of appearance.

The latter, on the other hand, appear to be imitative.

The **Atypical Tremors** are sometimes generalized, affecting all the body muscles and sometimes limited to a limb or part of a limb.

(1) When *generalized*, they are usually associated with other symptoms as a part of the clinical picture of the "shell-shock syndrome."

The facial expression seems to portray indescribable terror, and shares in the disorderly and irregular movements of the whole body. The tremor has been justly considered (Ballet) as the "expression mimicking fear." There are also circulatory

disturbances (tachycardia, flushing or pallor of the face), secretory changes (perspiratory, lacrymal, small salivary secretion, discharge of urine), all of which with the tremor are manifestations of the emotion (Henry Meige).

In the generalized tremors the patient is shaken by vibratory oscillations of variable intensity, sometimes fine and limited to the extremities, more often wide and irregular; in the latter there are extreme oscillations of the limbs reminiscent of chorea gravis, accompanied by clonic movements of the muscles of the face and neck. The patient cannot stand upright nor walk and has to be carried on a stretcher. On the slightest touch or examination (testing the reflexes, raising the bedclothes, or even the arrival of the doctor on the scene), these illogical and paradoxical movements become more violent and sometimes reach a surprising intensity. Any sudden noise, such as slamming the door, or particularly the sound of cannon, distant though it may be, may determine the occurrence of paroxysmal crises.

In one case in our hospital at Villejuif (February, 1915) presenting all the classical signs of Charcot's "grande névrose trémulante" following on the explosion of a shell, the paroxysmal aggravation of the tremor was particularly well marked. Every morning, on the arrival of the physician in the ward, the patient exhibited a pronounced accentuation of the tremor lasting several minutes, accompanied by dyspnœic symptoms. This gradually passed off, and the amplitude and intensity of the tremor regained its former level.

In one of Meige's patients, a sudden noise, sharp explosion, or visit to the trenches would bring on real motor crises with wide and generalized tremor so severe as to cause loss of equilibrium; then this severe shaking would subside gradually and the tremor regain its normal intensity. Ordinary testing of the reflexes was equally effective in causing this exacerbation.

In these paroxysms it is not, properly speaking, a question of tremor, but rather of sudden trepidation (Henry Meige): "The motor reaction which they represent is due to a brusque contraction of all the flexor muscles. The arms are closely applied to the chest, the forearms are bent on the arms, the fingers close up, the thighs flex on the trunk and the legs on the thighs. The trunk is bent forwards with few exceptions; the shoulders are raised and the head lowered. The facial muscles also share in this abrupt contraction; the eyelids close, the teeth are closely approximated, and the lips are pressed tightly together. In brief, the individual responds to the emotional shock by a reflex action of generalized defence which has the effect of hunching up the body and reducing its surface to a minimum. This phenomenon of retraction is seen in every scale of animal life from the amœba upwards, when sudden danger is imminent. It is followed by a more or less abrupt and complete relaxation. It is sometimes repeated several times in succession with first increasing, then decreasing intensity."

Lastly, in certain cases one frequently finds a distinct psychopathic condition, which Henry Meige has analysed and described under the name of *tremophobia* (fear of tremor). This is not rare in patients who exhibit tremor of the head or limbs. "Tremophobia partakes of all the characteristics of an obsession; it is similar to the ereutophobia (fear of blushing) of Pitres and Régis. It is seen in individuals with predisposition, especially those of generally unhealthy constitution. Blushing and tremor are, indeed, only reflex manifestations of the emotional condition. Just as the blushing of ereutophobia increases parallel with the fear, so the tremor of tremophobia is augmented by the fear of tremor. The physical phenomenon produces the obsession which, in its turn, increases the somatic reaction; the exaggeration of the latter again reacts on the mental disorder; thus a vicious circle of reciprocal

psycho-physical reactions is established, ending in a true obsessional disease " (Henry Meige).

(2) When *limited*, they are confined to the upper or lower limbs on one or both sides or to part of a limb.

The **Typical Tremors** resemble, either by the actual character of the tremor or by associated signs, the definite tremorous disorders which are well known and classified in neuropathology. In distinction to the first group (atypical tremors), which may be considered as products of the individual's own imagination, these typical tremors seem to be imitative, of course more or less unconsciously, except in cases of real malingering.

They may appear in various forms :

The pseudo-Parkinsonian type, in which the tremor is generalized, rapid, and regular, and predominates in the extremities, especially in the arms. The resemblance to Paralysis Agitans is sometimes so close that the two may be confused on the first examination and the diagnosis has to be gone into very fully.

Sometimes this form of tremor is localized to a part of the body only or to a single limb (hemi-Parkinsonian type).

The disseminated sclerosis type, in which the oscillations are of greater amplitude than in the preceding form. They are much accelerated by voluntary movements, and especially when the hand reaches out to grasp some object. However, the resemblance to disseminated sclerosis is only remote and will not bear careful examination.

In other and rarer cases the fine, rapid tremor, localized in the extremities, may resemble that of exophthalmic goitre, that of progressive general paralysis, or that of toxic (alcoholic) tremor, etc.

When the character of the tremor resembles that of exophthalmic goitre, and when, in addition, there is slight acceleration of the pulse-rate (sometimes of

emotional origin), the distinction between this condition and the abortive forms of Graves' Disease becomes extremely difficult to draw.

The important part attributed to emotion and physical or mental shock of every kind in the pathogenesis of exophthalmic goitre has long been fully described in the literature. Instances which apparently confirm this idea have been observed and published ever since the commencement of the campaign.

In our opinion, this pathogenesis is far from proven and most of the observations on the subject deserve careful scrutiny. Without wishing to labour the point, we will only say that we have not seen a single case of Graves' Disease in which close investigation has clearly established a definite connection between the trauma or emotional shock and the actual onset of the symptoms. It appears more logical to suppose that, as in many other affections of unknown pathogenesis but indisputable organic nature, the emotion or trauma only plays the part of bringing to light a latent disorder, just as a blow on the knee or hip reveals for the first time a swelling or hip-disease.

This question of the causal connection between trauma and certain pathological conditions (tubercle, syphilis, etc.) has a wide and practical importance at the present time ; in so far as it concerns nervous diseases it merits detailed consideration.

In the *pseudo-cerebellar type* the irregular movements of walking, imperfect equilibrium and ataxia more or less resemble the cerebellar syndrome.

Pseudo-choreiform types.—These are far from rare, and resemble the different clinical aspects of the various forms of chorea :—chorea gravis, with irregular movements of all the limbs, the head, the neck and also the facial muscles : Sydenham's chorea and rhythmical or saltatory chorea. All these types are specially characterized by affections of the gait. They are described in detail in the chapter dealing with this subject (p.753).

§ 2. The Tics

Tonic tic (postural tic of Meige and Feindel) may be observed as a result of wounds or nervous shock in war ; but it is much less common than the clonic tics or spasmodic movements which make their appearance under the same conditions as the tremors, *i.e.* after nervous shock, the result of explosion of a shell near at hand or at a distance. Like the tremors, they easily and quickly yield to psychotherapy when treated at a time as near as possible to their onset.

They are usually observed in and around the head : *e.g.* clonic contractions of the neck (sterno-mastoid, trapezius, platysma) or of the head, movements of affirmation or negation, palpebral or facial spasms on one or both sides or movements of elevation of one or both shoulders.

Evolution and Prognosis.—The evolution, duration and termination of these phenomena—as in all the psychoneuroses—are so variable that we cannot give any general description either of their evolution or prognosis. They are cases which show great variation according to the course pursued during their successive transference from one to another of the various hospitals and institutions.

Though we sometimes have to deal with old cases in which after long disappearance of the tic, this has again come to light owing to the fatigue or the emotions of the campaign, we more often come across these symptoms appearing for the first time.

In exceptional cases the affection may be improved or even disappear after a simple period of rest behind the lines ; but the majority of these cases of tremor or clonic movements, when left to themselves, seem to persist almost indefinitely. We know of many instances in the general hospitals, the special departments and elsewhere, in which a tremor has persisted for many months, often over a year ; there are patients who have had a tremor ever since the

battle of the Marne. Everything depends on the time of treatment and the way in which they are interpreted by the physician who examines them.

With regard to **Relapses**, we cannot at present make any definite statement. The observations published by various authors, ourselves included, are necessarily incomplete on account of the impossibility of following up the cases for a sufficient length of time.

In any case, it is certain that substantial reservations must be made in these individuals as regards their future fitness for military service. If the affection has suddenly appeared in a man previously sound and has rapidly disappeared under treatment, one may justifiably consider the cure as likely to be permanent and expect the soldier to be able to take his place in the trenches after a certain period of rest. If, on the other hand, we are dealing with a particularly nervous, emotional individual with a previous history of other neuropathic symptoms, the prognosis as regards his military usefulness in the front-line is poor.

The **Nature** of these affections, especially that of the tremors, has been much discussed.

At a recent meeting of the Paris Neurological Society, Babinski, in his paper on " The Character and Methods of Dealing with the So-called Functional Motor Disorders," raised the following point for discussion :

" In the neurology of the war, many cases of tremor have been observed, often following shell-shock. They present none of the characteristics which belong to those organic affections with symptoms of tremor, and, moreover, are uninfluenced by psychotherapy. Some of the members of the Society have specially studied these tremors and may be able to specify their nature and the methods which should be adopted in dealing with them."

Henry Meige was of opinion that they might be of emotional or concussion origin. Emotional

tremor—the manifestation of the emotion—is signalized by oscillations very variable in frequency and amplitude. . . . Generally transitory, and disappearing with the other elements of the emotional syndrome, it may persist for a long time and even become permanent. In this case it must be admitted that some organic disturbance has been produced under the sole influence of the emotional shock. The intractability of the symptoms, their resistance to all treatment and every psycho-therapeutic method, confirms this standpoint.

In the concussion tremors, another factor is at work. The explosion acts mechanically by the disturbance and sudden change of pressure which it produces, not to mention the fact that the individual is violently thrown to the ground against a resistant body. This variety of tremor may thus very well depend on an organic lesion and seems to arise from a definite disturbance of the nervous apparatus.

Guillain also upheld this view of their pathogenesis.

We find it difficult to conceive of these tremors following nervous shock after an explosion being produced by an organic lesion.

We find a proof of our standpoint in the cure which is possible by means of the psycho-therapeutic measures employed at the correct time ; this cure is often rapid or immediate and lasting.

Though the fact that a nervous disorder yields rapidly or immediately to psychotherapy is an undeniable proof that it is not of organic nature, the reverse is not necessarily true. It is incorrect to ascribe the incurability of certain nervous manifestations to the supposition that they are caused by an organic lesion of the central nervous system. Is not the persistence of some pithiatic or hysterical disorders a fact known to all ? And in the large Parisian hospitals (Bicetre or Salpêtrière) one can find patients with indisputable hysterical affections, long-standing though they may be, the nature of which is obvious to all.

Although therapeutic measures here prove unsatisfactory and though the prognosis of the tremors and tics is the most unfavourable, are these sufficient reasons for considering them of an organic nature, fit cases for discharge from the army ? We think not, seeing that we have seen cases of our own, since sent back to active service, which have proved ultimately curable.

Differential Diagnosis.—The diagnosis of tremor and tic due to nervous shock raises the general question of the diagnosis of the classical nervous affections, on which we cannot dwell. A few rudimentary facts, culled from observations made during the war, merit consideration. In the differential diagnosis, importance must be attached to the following characteristics :

(1) *The Mode of Onset*, always or nearly always rapid and sudden, more or less directly connected with a nervous shock, an emotion, or the explosion of a projectile without external wound.

(2) *The General Character of the Abnormal Movements or Tremor*, which are generally arrhythmical and irregular in their intensity and development, usually exaggerated by voluntary movements (intention-tremor). As Meige states, the latter point is of little value. An important point is that the tremors, especially those of the limbs, are accompanied by a *permanent* contraction of the groups of muscles concerned : a contracture of the muscles of the neck in tremor of the head, contracture of the wrist and elbow in tremor of the upper extremity, etc. When during the course of treatment these contractures disappear for a few moments, the tremor quickly ceases. Thus when carrying out therapeutic measures, care must always be taken to obtain complete muscular relaxation by getting the patient to open the mouth and breathe deeply.

(3) *Associated or Accessory Symptoms*, of somatic or psychological order, are of great service in diag-

nosis; such are disproportionate efforts, manifestations of dyspnœa and emotional signs which may accompany interrogation or the first attempts at treatment, viz., passive movements of a contracted limb.

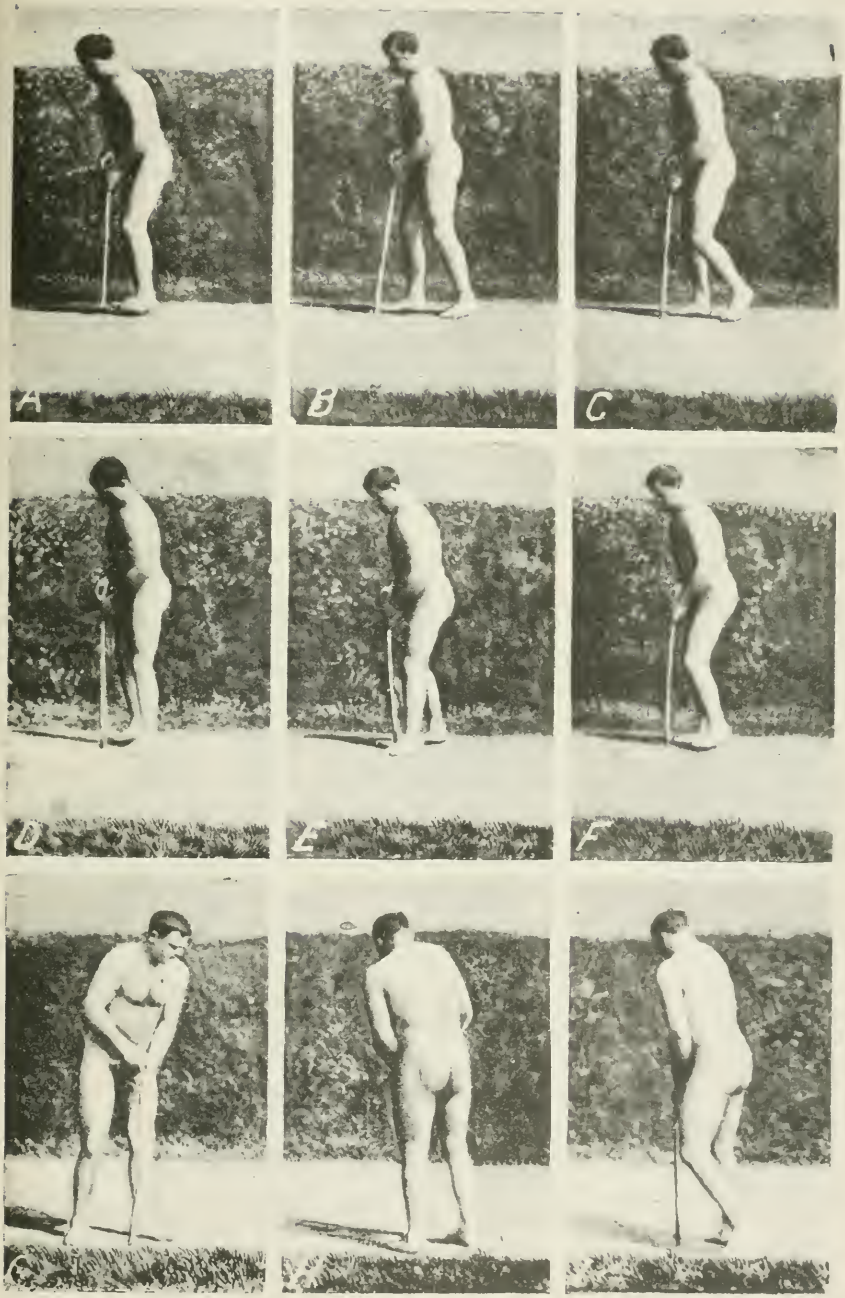
(4) The characteristic *Psychical Condition* of these patients, which causes them to attach too great importance to the disorder from which they are suffering; also the fact that they try, from the commencement of the examination, to distract the attention of the physician, to lead him along a false track by talking about subjective symptoms other than those for which they were sent to hospital; this renders the examination difficult, as these patients are usually loquacious. Frequently these first psychological impressions give the physician a clue to the diagnosis and indicate fairly clearly the true nature of the affection.

(5) *The Absence of any Somatic Sign* in respect of the cutaneous or tendon reflexes, *i.e.* the absence of any real modification such as abolition or exaggeration with epileptiform trepidation, showing a disturbance of the reflex arc. One too often finds that a physician not specially trained in neurology will attach importance to a certain degree of briskness of the tendon reflexes: "exaggerated reflexes, plantar hyperæsthesia" is often found on the case-sheets, signs which simply indicate a nervous excitability common in neuropathic subjects. Needless to say, such statements have a bad effect on the patient.

It is hardly necessary to mention that under no circumstances must a search for supposed stigmata of past hysteria be made, for this might entail the establishment of fresh neuropathic manifestations in patients so extremely sensitive to suggestion.

(6) *The Rapid or Immediate Curability* of the morbid condition, lastly, is of great value in differential diagnosis. Every case of tremor, tic, or choreiform movement which yields to psychotherapy

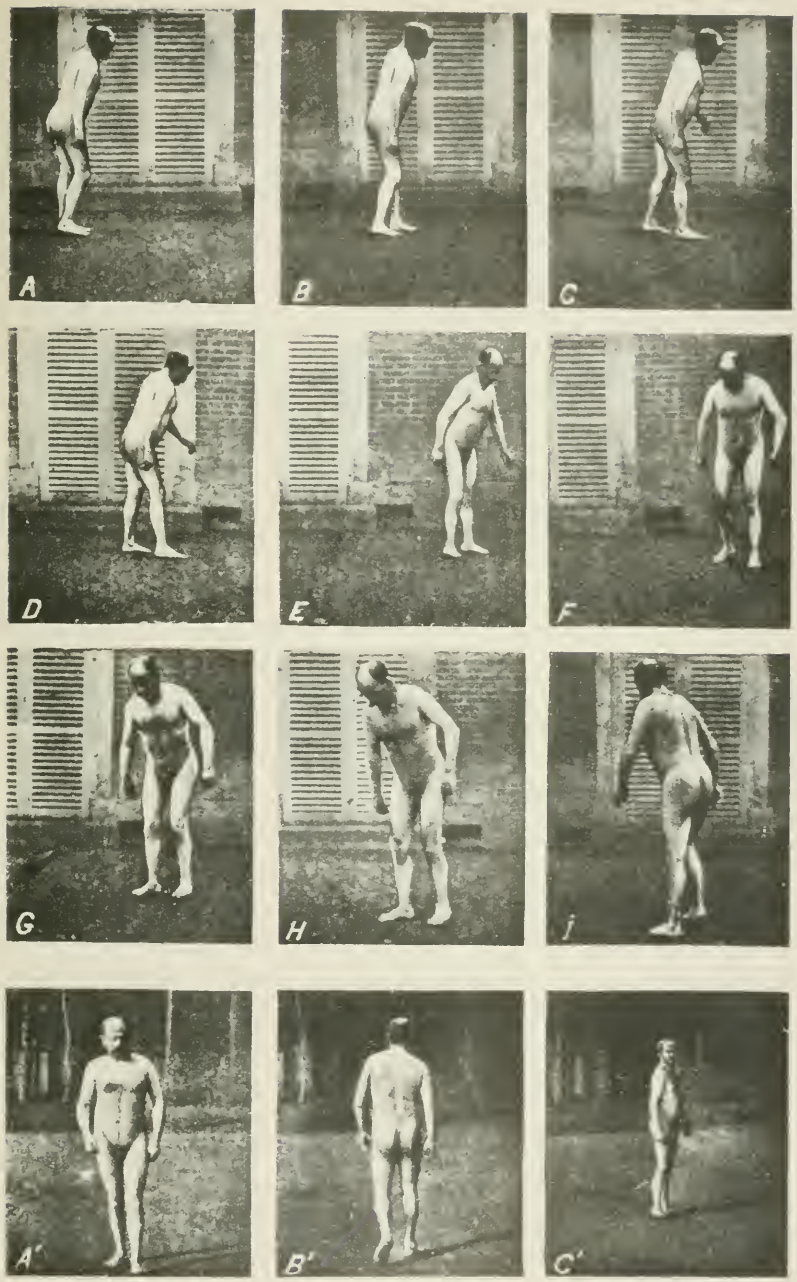
PLATE IX.



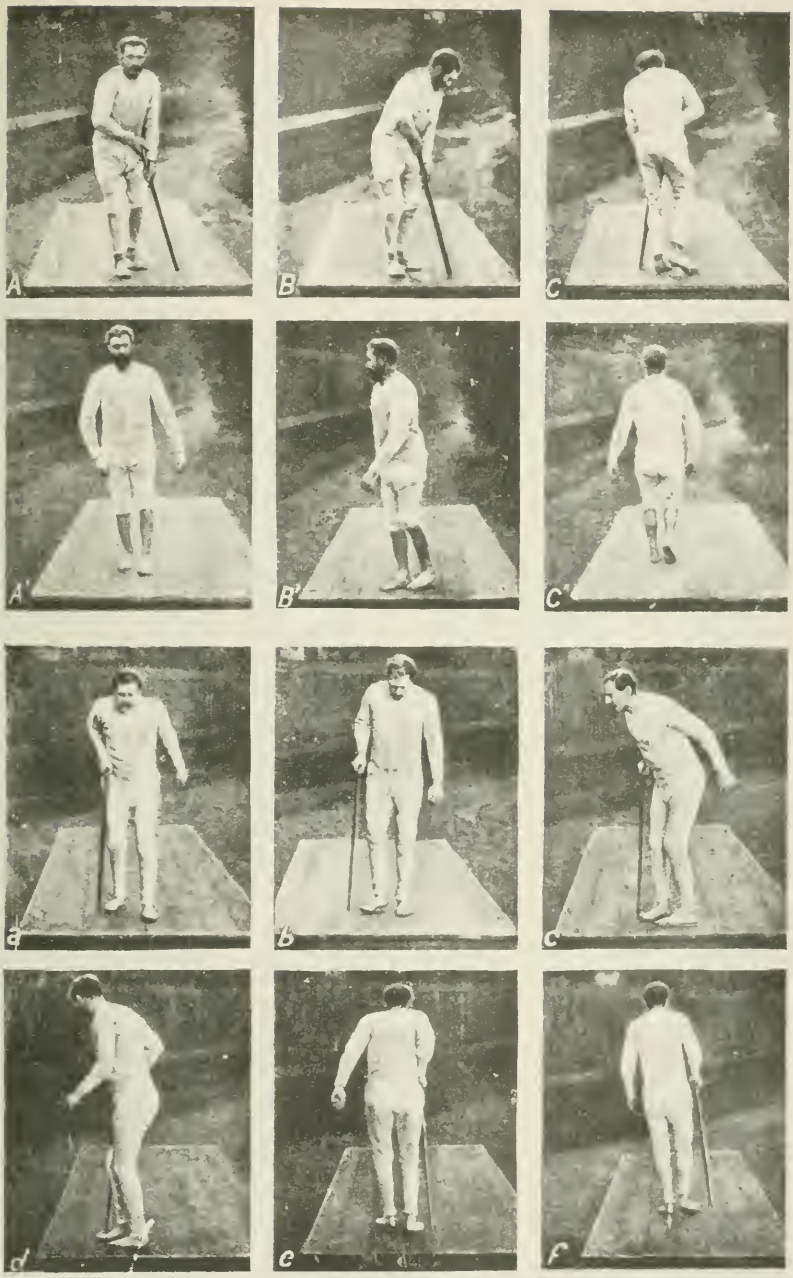
A-I.—Psychoneurotic disturbances of the gait: type seen in hip-disease. Tripod-walk.

A.-I.—Psychoneurotic disturbances of the gait with "bent back" of three weeks' duration.

K.-M.—The same patient, after one séance of psycho-electrical treatment, cured.



A.-I.—Functional paraplegia, spasmodic type, with slight camptocormia.
A', B', C'.—Cured.



A, B, C.—False sciatica (right side) of several months' duration.
A', B', C'.—The same patient four days later, cured.
A-F.—Functional monoplegia (right), following sciatica.

A-I.—Prolonged claudication due to false sciatica (left).

must be considered as a functional, hysterical affection.

These few points should always be borne in mind by the physician called to examine a psychoneurotic patient. They will help to reveal organic affections with tremor which, though rare in the neurology of war, may be met with. One may discover cases of family tremor which have accidentally been passed into the army, cases of true Sydenham's chorea in the younger soldiers or organic affections such as disseminated sclerosis or a cerebellar syndrome coming to light spontaneously during the course of the campaign. But in these cases the etiology is different, and the disorder only exceptionally follows a nervous shock due to explosion of a projectile. As a rule, the disease appears spontaneously. The general symptomatology of these conditions will enable them to be recognized, given a certain practical knowledge of neurology.

We have intentionally refrained from mentioning affections due to pure malingering, this question having been discussed in a separate chapter.

Chapter IV

PSYCHICAL DISTURBANCES OF SENSATION

These occupy an important place amongst the psycho-neurotic manifestations of war, both owing to their frequency and duration and to the functional impotence which they produce.

They may be divided into two main groups, according to the degree of frequency :

(1) *Psychopathic pains or* " *algias,*" subjective disorders of sensation which are extremely important owing to their common occurrence, duration and practical consequences from a military point of view.

(2) *The Anæsthesias* or objective disorders of sensation are much less important both from a semiological and practical standpoint.

§ 1. THE " ALGIAS "

Frequency.—These are very common. A large number of soldiers of all ages and conditions are found in the hospitals, field-ambulances, or orthopædic institutions, or are treated in the interior for pain and functional disturbance of a purely neuropathic character. Fortunately, since the Military Neurological Congress held at Doullens in January 1916 and owing to the work of the Under-Secretary of State and the Director-General of the Medical Services, a more effective method of filtration for these cases has been organized in the army.

To make ourselves quite clear, let us say that under the term *psycho-neuropathic pain* we include every pain, with or without loss of power, which

on clinical grounds or for therapeutic reasons, for example, cure by psychotherapy, does not appear to be caused by an organic lesion.

Of all the varieties of pain observed, that localized in the lower limbs and causing claudication is the most common. It produces the most rapid and certain demobilization and return to the field-ambulance, casualty clearing-station, or base hospital of the individual in question. It is also the most easily simulated and the most difficult to control.

The Causes are variable. They sometimes appear to arise spontaneously without any apparent cause, or follow some very slight disorder such as a minor attack of rheumatism, a febrile condition or some infection. They more often have some definite causal condition, *e.g.*:

(*a*) Any form of trauma, shock, blow from the butt of a rifle, fall from a motor-cycle, or temporary burial in a collapsed trench, without any external sign of injury being observed.

(*b*) A slight gunshot, shell, or shrapnel wound, a small foreign body left for a time in the superficial tissues, shown by skiagrams, and to which the patient's attention has been specially directed.

(*c*) Some previous organic lesion, long since cured: patients who have had true organic sciatica, slight rheumatoid arthritis of the hip-joint, knee, or foot, rheumatoid or traumatic lumbago.

(*d*) Lastly, more severe lesions such as frost-bitten feet and "neuritis," which threaten to persist indefinitely if there is no intervention of a radical nature at the right time (G. Roussy and R. Leroux).

The previous condition of the patient appears to be of little importance. These affections may come on at any age, being seen both in soldiers of the younger classes and in the older men on home service; they are found in individuals without

previous neuropathic taint and in predisposed and alcoholic subjects.

The Clinical Types show infinite variation, but in general they reproduce the classical syndromes in which pain is the important factor. These may be vicious antalgic postures such as curvature or scoliosis, or functional impotence as seen in the various forms of claudication. They may take the form of more or less severe pain without apparent deformity, but localized in clearly defined regions to which the patient constantly directs attention (hysterical or neurasthenic topo-algias). Lastly, they may be cases of various visceral pains—gastric, vesical, renal, etc.—the diagnosis of which is often far from simple.

Thus it is difficult to give a general description of the clinical aspect of a varied selection of pains such as are of almost daily observation in the military neurological services.

In order to adopt some general scheme for such a vast group, we will adopt the following classification :

(1) *Algias of the lower limbs* (quite the most frequent).

(2) *Algias of the upper limbs* (rarer).

(3) *Algias of the trunk and spine* (not uncommon).

(4) *Algias of the neck and head, severe headache.*

(5) *Visceral algias.*

Psycho-neuropathic Algias in the Lower Extremities.—The number of these is so considerable that it is no exaggeration to say that there are very few specialized or non-specialized departments, orthopædic or convalescent institutions, where there are not a few soldiers suffering from pain of a purely pithiatic nature.

Although hardly systematic, but nevertheless useful from the point of view of differential diagnosis, we may arrange them in three principal groups according to their resemblance to some painful organic affection of the lower extremities.

Pseudo-Sciatic Type (Roussy and Grutzhaendler). —This is the hysterical sciatica of the classical authors and is of great frequency.

The *Onset* of these false sciaticas is only occasionally spontaneous. They sometimes appear as the result of a trauma—a blow on the knee or leg, slight superficial wound, etc.—and entirely resemble the general type of hystero-traumatic disorders. But more frequently it is after more or less vague discomfort of a rheumatic or rheumatoid nature in one or both limbs that these pains make their appearance and cause limping and complaints of pain, lasting weeks or months. A slight organic lesion or sciatica coming on at regular intervals is the common ground on which these psycho-neuropathic symptoms take root. These psychical affections may sometimes appear quite genuine, unconscious, sometimes subconscious or exaggerated (re-exaggerated sciatica) or prolonged (prolongation of sciatica, with habit-claudication), or even wholly created for some easily discernible object (simulated sciatica). But we must humbly own that the physician himself often plays a part in the appearance and fixation of these affections; for the mistaken diagnoses written up in the case-sheets, suggestions for change of military status, and the many external stimuli around the patient only reinforce the ideas they have of their disorder.

The distinction between these three conditions—real psychical disorders, exaggeration and simulation —is often difficult and sometimes impossible to draw; only such proofs as confession or flagrant mistakes in conduct will justify the conclusion that a pain or functional impotence is simply a case of malingering.

The Clinical Aspect of patients with false sciatica is sometimes so similar to that of true sciatica as to be mistaken for it. These are individuals with pain in one leg, occasionally but rarely in both, and who limp. We must therefore discuss the diagnostic value of the pain and its result—the claudication.

(1) *The pain* gives little information as regards

the diagnosis, as its verification is difficult. The patient complains of pain along the distribution of the sciatic nerve, in the buttock, thigh, or leg, often also around the sacro-iliac synchondrosis. Deep pressure (Valleix) evokes or seems to evoke the pain. It is sometimes very acute, preventing sleep; Lasègue's and Bonnet's signs are positive, but in this connection one must take into consideration the degree of education created in the patient as a result of the many examinations that have been made. Sometimes, however, the site of the pain is more paradoxical. The patient complains of pain in a diffuse form in the groin, the lower part of the thigh, the knee or lumbar region. Such indications point in favour of false sciatica. Objective disturbances of sensation are much more common than in organic sciatica. One may find anæsthesia of the "hysterical type" in large bands or patches, sometimes affecting the whole of the lower extremity (signs in favour of a pseudo-algia). In rarer instances, there are parallel bands of hypæsthesia or anæsthesia more or less resembling the distribution of a nerve-root. Lastly, it may take the form of hyperæsthesia of the skin in the lumbar region, thigh, or sole, intensified by walking. All these objective disturbances of sensation may, we think, be explained by previous medical examinations. If tests of sensation are carried out by Babinski's method (p. 796), one never discovers these disorders of sensation in patients who have not undergone complete examination; and in those cases that do exhibit them they are always found to disappear on the application of the faradic current.

(2) *Claudication* in patients suffering from false sciatica is often very pronounced, usually more so than in true sciatica. Progression is effected by hopping along with the lower limb maintained contracted either in extension or flexion. The patient gets along with both hands holding on to a stick, which he holds in front of him and at each step he

presses the handle against the abdomen (tripod-gait). As a rule it is only the point of the foot or the toes alone which touch the ground, or the foot may lie flat but the toes are maintained in dorsal hyperflexion.

Running is naturally impossible, the patient refusing to attempt it. In the standing position the patient holds himself hunched up on the sound limb and resists all attempts to correct this attitude; thus the gluteal fold on the affected side is diminished or obliterated and the natal cleft is deviated from the affected limb. In other cases more unusual postures are adopted: the trunk bent forwards or laterally from the healthy side, pelvis tilted to the affected side with false shortening of the limb and compensatory scoliosis of various types.

During attempts to separate the thighs, one can nearly always observe a contracture of the adductors, for which there is no reason. These patients resist all attempts at passive movement of the painful and often contracted limb, both those which entail stretching of the sciatic nerve and those which make for its relaxation. At this point of the examination there appear certain signs almost always observed in the psychoneuroses:—dyspnœa and contraction of the diaphragm (efforts which ought to invoke the pain), screaming and shouting, an outburst of weeping or a true convulsive attack. When the patient, lying down, is told to get up quickly, one may observe in the less severe cases that he raises himself upon the painful limb just as well as on the unaffected.

All the somatic signs of true sciatica will be completely absent: all the cutaneous and tendon reflexes are normal. They are occasionally rather brisker than on the normal side; there is no hypotonicity. A diffuse muscular atrophy is occasionally observed in cases of very long duration and certain vasomotor symptoms (cyanosis, excessive sweating); these can be explained by the prolonged immobilization.

Such are the clinical signs which must be used to distinguish false sciatica. The differential diagnosis is sometimes admittedly difficult; to decide in the case of a patient who is stated to have had at one time true sciatica, which are the true symptoms and which are simply superadded or exaggerated is often no easy task. Be this as it may, it seems that in case of doubt it is always preferable to decide in favour of false sciatica and to treat it as such: psychotherapeutic measures, if not successful, will at any rate afford the clue to the diagnosis.

Pseudo-coxalgic Type (Roussy and Grutzhaendler). —This is the hysterical "coxalgia" of the literature, and is almost as common as the last variety. In these cases it is around the hip and the hip alone that the pain is localized. The claudication resembles that of hip-disease. The limb is drawn up, the knee in extension or partial flexion. At every step the trunk is bent sharply forwards on the affected side. When walking, the movements of flexion and extension at the hip-joint are limited or absent. When an attempt is made to passively move the joint, defensive contractions are observed; sometimes one is able to take the patient by surprise by making a movement opposite to that which we wish to obtain:— extension of the thigh on the pelvis when an attempt is being made to flex it. Signs of shortening, of pain produced by percussing the knee with the leg in extension, external rotation—all these may be present as in true hip-disease. Thus the diagnosis is sometimes a matter of great difficulty, and a surgeon has to be called in to make a thorough examination of the joint, for which an anæsthetic is often necessary or else a skiagram has to be taken.

The therapeutic criterion—cure by psychotherapy —has the greatest value.

The Pseudo-cruritic Type is much less common. It generally takes the form of a contracture at the

hip-joint and is mistaken for crural neuralgia or the onset of hip-disease.

The same considerations hold in **pseudo-arthritis** of the other joints of the lower limb—of the knee (*gonalgia*), of the instep (*tarsalgia*), or of the heel (*pseudo-talalgia*), which are often labelled sciatica.

All these also produce claudication, often in a marked degree; their differential diagnosis entails the same methods of examination as in cases of coxo-femoral arthritis.

Pseudo-neuritic Type.—Under this heading we include the various pains, localized in one or both limbs, following an infectious disease or a simple gastric disturbance. These are accompanied by vague discomfort, and have been put down as due to "neuritis" of the lower limbs.

Now, influenced by this diagnosis, the patients will often continue to complain of pain in the limbs for a great length of time. In this way disorders of the gait are caused without the observer being able to discover a single sign indicating the existence of any actual neuritic lesion.

We can include in this group a large number of disturbances of the gait due to so-called neuritis following on frost-bite (Roussy and Leroux).

This usually happens in the case of soldiers sent back from the regiment or from an orthopædic institution to hospital, with the following diagnoses: nervous symptoms, neuritis or neuritic phenomena, œdema, hyperæsthesia, following on previous frost-bite of the feet. The original cause (frost-bite) generally dates back several months, often over a year.

The clinical symptoms consist of disturbances of the gait: more or less unusual limping caused by a paradoxical vicious posture of one or both of the feet, sometimes even of the legs, with pain which necessitates the retention in or return to hospital of these patients.

On examination, one or both of the feet will be found in a vicious posture, sometimes permanent and sometimes only existing when the patient walks. He does this on the external border of the foot, chiefly on the heel, the toes dorsally flexed or the foot even in true equino-varus position. This is the chief sign and the one which immediately attracts attention.

On closer observation vaso-motor changes are observed—bright purplish colour of the skin of the foot, prolonged pallor after pressure on one spot, lowering of temperature of the skin (2°-3°) reaching a certain distance up the leg, and sweating. It must be noticed that it is a question of segments of the limb long immobilized. These vaso-motor phenomena are far from common, we may even say rare. The same can be said of œdema, which, although figuring on the case-sheet, is often transitory, hardly perceptible, or absent. The patients themselves, however, make a good deal of this symptom, which is barely obvious to the naked eye.

One other important feature makes the clinical picture complete : this is the presence of pain—spontaneous or occasioned by walking. This may be acute and described as burning or pricking, or sometimes only consisting of simple hyperæsthesia of the sole of the foot. These aches and pains last for weeks or months ; they render walking well-nigh impossible (pronounced lameness, walking with a stick or a crutch). They cause and maintain the vicious posture.

Now, in cases of this nature, a minute examination shows that there are no organic disorders at work sufficiently pronounced to justify such functional incapacity. There is no evidence whatever which could point at this time to any lesion of the peripheral nerves or the vessels. On the other hand, all cases of such pain and vicious posture yield extremely rapidly and in a most surprising manner to psychotherapeutic and electrical methods undertaken from the time of the patient's admission. Further, as the

patient regains the normal use of his feet or his contracted and painful limbs, the œdema and the vasomotor or thermal disturbances will disappear *pari passu* with the vicious posture.

Algias of the Upper Extremities.—These are much less common than the last group. This is easily understood, given the lesser degree of functional impotence of which they are capable. They are usually unilateral, and only exceptionally bilateral.

Like those of the lower limbs, the pains may resemble either neuritic or articular pain.

In the **pseudo-neuritic** form the pain is vague and diffused over the limb or a segment of it, occasionally localized to the distribution of a large nerve trunk, and has originated in a shock, a superficial wound, or it may be of quasi-spontaneous origin. The patient gives little precise information as to its nature, intensity and distribution, but chiefly dwells on the impossibility he experiences of carrying his rifle, lifting a weight, or bearing his kit.

What is more frequently seen, especially in men on home-service, is the amplification of some real pain, in the distribution of the median or ulnar nerve for example, a manifestation in which exaggeration seems to take a considerable share.

These pains may exist alone or quickly become accompanied by other hysterical disorders—anæsthesia or functional loss of power due to paralysis or contracture.

In the **pseudo-arthritic** form—rare in the elbow and wrist but common in the shoulder—very much the same clinical picture is found as in articular pain in the lower limbs. The patient has had pain for some months around the joint, which he immobilizes more or less completely, often even resisting all attempts at examination.

Especially in the case of the shoulder-joint, differ-

ential diagnosis is often very difficult and necessitates thorough examination (under an anæsthetic, or by skiagrams); this the more, as pithiatic phenomena are often grafted onto an organic condition—dry arthritis, periarthritis, etc. The therapeutic measures still in common use, the faulty diagnoses written up on the case-sheets, the psychical condition of the patient, surrounded by possible sources of "infection," all rapidly make for the fixation of the pain.

Algias of the Trunk (spine, thorax, etc.).—Much the most common of these are painful affections of the vertebral column which, in order of frequency, stand next to those of the lower limb. Such are the various forms of pain which engender deformities of the trunk and the vertebral column (scoliosis or curvature).

The Rhachialgias of neuropathic order consist of vague pains, generalized throughout a region (cervical, dorsal, or lumbar) or localized around one or several of the spinous processes, thus resembling the clinical aspect of Pott's Disease. They rapidly produce deformity and vicious postures which, though at first transitory, inconstant and easily reduced, gradually become permanent and irreducible.

These, then, are at their onset cases of pseado-spondylitis with the element of pain predominant, spondylitis with affections of the gait, impossibility of lying on the back and of moving the vertebral column. The patient walks with a slight forward inclination. He can, however, hold himself upright, though with great difficulty; in all recent cases reduction is easy without using forcible methods.

Some shock, the falling in of a trench, a beam or portion of a dug-out, or the explosion of a shell close at hand has been the origin of the affection, preceding it by a few hours or days. The patient has almost always lost consciousness; he complains of pain in the back as soon as he comes to himself.

Such are the cases which at the commencement of the war were only too often erroneously put down as "hæmatomyelia," "medullary lesion," or "medullary disturbance." However, these patients, in the absence of any grave medullary lesion, when reassured as to their condition, always recover easily and rapidly if one only intervenes in time.

Even in old-standing cases a cure can be obtained, especially if they are sent on to the special Military Neurological Centres.

At a later stage contractures, causing permanent vicious postures, are added to the symptom of pain. The spondylitis is replaced by a pseudo-spondylosis; the symptoms, indeed, correspond to an ankylosis of the vertebral column (scoliosis, kyphosis, lordosis). This is not the case, however. It is the contracture of the spinal muscles which immobilize it, as is proved by skiagrams, examination under anæsthesia and cure by various therapeutic measures. These various types of contracture or camptocormia (Souques) have been described in detail in Chapter I. (p.737).

The first type—painful spondylitis—representing the early phase of the malady, is seen specially in branches of the medical service at the front, while the second—pseudo-spondylosis—is the form observed in the various neurological centres in the interior, where examples are now to be found in large numbers.

The Lumbalgias or Pseudo-lumbago.—This fairly common form is only a variety of the last condition. It has the same etiology: falling in of a trench, shock, or severe trauma. It has the same clinical aspect: immobilization of the spine in the dorso-lumbar region, with muscular contracture, pain, curvature and disorders of the gait, the patient walking with short, abbreviated steps. It deserves separate consideration on account of its frequency and its resemblance to true traumatic or rheumatoid

lumbago, from which it is often difficult to differentiate. This difficulty is increased by the fact that, as in sciatica, it is often a case of simple prolongation or exaggeration of a painful affection—an organic lesion with a psychical element superadded.

Such cases are transferred to the specialized departments for diagnosis and investigation labelled hystero-traumatism, traumatic lumbago, lumbar pain, etc.

We have still to mention the **Algias of the Thorax**—sharp pain in the side, pleurodynia and false intercostal neuralgia—spontaneous or traumatic, often accompanied by dyspnœa, affections for which no organic cause is revealed by thorough examination of the respiratory apparatus and thoracic wall or by skiagrams. According to our personal experience, they appear rare and are of little importance.

The same holds for the **abdominal algias**—false appendicitis, pain in old hernia scars, etc., which, as psychoneuroses of war, exhibit only the characters described in the classical treatises.

Algia of the Neck and Head.—Pain in the nape of the neck may be met with. It rarely appears as the sole symptom, but is generally accompanied by spasm of the muscles of the neck—**tonic or clonic torticollis**—which has already been described.

Severe headaches form an important group of the nervous manifestations incidental to the war. But various types have to be distinguished, all of which do not come within the range of the affections with which we are concerned.

(1) *Headache of true hysterical or pithiatic type* appears very exceptional, if we include only under this heading the inorganic affections which are capable of rapid and complete cure by a few séances of psychotherapy.

(2) *Neurasthenic or psychasthenic headache,* on the other hand, is a frequent component of the symptom-

atic picture of neurasthenia. There is no special characteristic as regards the psychoneuroses of war; even when it only exhibits its ordinary degree of persistence, it necessitates prolonged treatment and transference of the patient to one of the hospitals in the interior.

(3) *Headache due to bombardment* is extremely common; it forms some part of the delayed concussion syndrome, and one may safely say that nearly all patients subjected to the explosion of a shell near them suffer from it, sometimes for a long period after the original shock. It is persistent in the occipital or frontal regions, little affected by analgesics; sometimes it is improved by lumbar-puncture, which may reveal slight lymphocytosis and increased pressure. This form of headache hardly comes within our scope, for it appears to depend on an organic change—vascular or in the cerebro-spinal fluid—and is not improved by psychotherapy.

(4) *Headache in patients who have previously been trephined* forms a last, important group owing to its frequency and practical bearing. Pierre Marie has just recently drawn attention to the importance of this sequel of trephining. But here again we have to deal with an affection arising from a previous organic lesion, to which a certain degree of exaggeration or persistence may be superadded.

Visceral Algias.—These are fairly rare as true neuropathic or hysterical manifestations following a trauma; they more commonly form part of the neurasthenic or psychasthenic syndrome:—*pain in the testicles* following a wound or castration, *cystalgia* in patients suffering from shock, *gastralgia* accompanied by vomiting in "gastropathic" subjects, *precordial pain and dyspnœa* common in patients who have been exposed to asphyxiating gas.

These are usually accompanied by neuropathic visceral affections which are described in the chapter devoted to this subject (Chapter VIII.).

Course and Prognosis of the Algias

The duration of neuropathic pains is extremely variable. Many factors modify their course, such as variations in diagnosis, different methods of treatment, evacuation at the right time or discharge from the army. The war has shown that these pains, like other hysterical symptoms, are conditions in which the patient is to a great extent at the mercy of his surroundings.

Our medical service, therefore, has set itself to make the environment suitable and to segregate into the Special Departments all soldiers who claim to suffer from pain of long duration.

To recognize the real cause of the pain, to determine the extent of psychical factors inducing exaggeration, prolongation, or simulation, to avoid the suggestion of a complaint to these patients but rather to reassure them, to give them, in fine, a motive and an opportunity of recovering—such are the objects all our medical men ought constantly to have in view in dealing with the psychoneuroses of war.

Further, we do not hesitate to affirm that every inorganic, functional pain can be and must be cured (or at any rate ought to be). The question of duration and prognosis thus depends on the diagnosis that is made and the therapeutic measures that are employed.

DIFFERENTIAL DIAGNOSIS OF THE ALGIAS

Diagnosis of these conditions is sometimes a matter of ease, at other times of considerable difficulty.

It is easily established when one has to deal with a patient presenting other signs of a neurotic order, such as convulsive crises, tremor, etc., when the pain is directly associated with trauma or when it appears as a sign of relapse in a patient with a clearly neuropathic history. On the other hand it becomes no easy matter when the pain or its results (functional

impotence) are the first signs observed or the only symptom. Diagnosis becomes increasingly difficult in those cases in which the psychical element is superadded to an organic lesion. It then becomes a question of gradation, degree, and experience.

In discussing the various clinical types, we have endeavoured to lay stress on the signs which assist in differential diagnosis, such, for example, as distinguish the pseudo-sciaticas from true sciatica and pseudo-coxalgia from disease of the hip-joint.

With regard to the diagnostic value of the various symptoms, it must be realized that pain itself is hardly sufficient as a diagnosis. There is, indeed, no positive and certain sign of the pain; and it is impossible to declare that a patient who complains of pain is not really suffering, except, of course, in cases of pure malingering or discovery of intentional deceit.

A basis must therefore be made on a certain number of signs, which, when grouped together and considered comparatively, acquire a real and definite value in diagnosis. The principal are these :—

(1) *Distribution of the Pain.*—It is usually paradoxical in its nature and erratic in its localization, *e.g.* pain in the hand, with complete functional impotence of the entire arm; lack of definite connection between the trauma and the pain, *e.g.* trauma of the left flank causing pseudo-coxalgia of the same side or of the opposite side, etc. These pains do not attack the anatomical distribution of peripheral nerves or nerve plexuses, and are not accompanied by painful areas along the course of the nerve or around the articulation; they are mobile and frequently change their locality. Pressure on a painful area does not produce dilatation of the pupil.

Their results are inconstant, sometimes being shown by slight loss of power or claudication, sometimes by very pronounced motor disturbances. Frequently in the course of a somewhat prolonged examination, or after repeated examinations, they

may be found to vary considerably in intensity from day to day or during a single examination.

(2) *Associated Signs noted during Examination.*—In addition to the symptoms complained of by the patient—the pain and its immediate effects, such as claudication, functional impotence, permanent or transitory vicious postures—there is a whole series of accessory or associated signs which come to light during the examination of the affected limb or region, which often give a clue to the diagnosis. In general they are much the same as those described in the previous chapter dealing with tremors, *i.e.* :—

(*a*) Respiratory manifestations (the dyspnœa of exertion or emotional dyspnœa) and the resistance shown by the patient to attempts at examination or passive movement. The patients complaining of pain in a limb or a joint make extraordinary efforts to immobilize the limb, efforts which are calculated to increase the pain, though the latter does not occur.

(*b*) The psychological condition of the patient, who is often uncommunicative, unwilling to look the observer in the face, always ready to turn the conversation, to switch the medical inquiry to other lines which shall not run in the intended direction.

(*c*) The occurrence of fresh nervous phenomena :— tremor, a modified or real convulsive crisis, an outburst of weeping, sweating, etc.

(3) *The absence of somatic signs* is, of course, of great value : absence of any real modification of the reflexes, of muscular atrophy, ankylosis, vaso-motor disorders and of electrical changes (in quite recent cases or slight motor affections). If, on the other hand, the motor affection has become predominant or permanent, then the prolonged immobilization will produce fibro-tendinous retractions, diffuse amyotrophy, decreased electrical excitability, and vaso-motor symptoms. All these have been described and their diagnostic value discussed in Chapter I.

(4) *Therapeutic tests and their results* are naturally the best criterion. The rapid disappearance of pain of long duration as a result of a single séance or a few séances of psychotherapy proves its neuropathic (inorganic) nature, especially when the cure is acknowledged and admitted by the patient and when it is accompanied by a simultaneous disappearance of the vicious posture or functional loss of power.

Equal diagnostic value is attached to the results of the opposite method of treatment—injections of morphia, cocaine, or stovaine—which cause only slight or no amelioration of the pain and may even increase it.

Lastly, we need hardly say that there should be no hesitation—especially in cases of neuropathic affections combined with an organic element—in resorting on every suitable occasion to positive methods of examination such as lumbar-puncture, examination under X-rays or skiagrams, and examination under chloroform or spinal anæsthesia. Such methods will sometimes reveal, for example, some slight osseous or vertebral lesion which is the origin of all the symptoms: they will always react favourably on the mind of the patient if care is taken to avoid using any imprudent expressions in his hearing.

§ 2. ANÆSTHESIA, ANALGESIA, HYPERÆSTHESIA, ETC.

Modifications of objective sensation are much less important than those of subjective sensation discussed in the preceding section.

Not being perceived by the patient, they produce no loss of function. This is the chief characteristic ascribed by the classical authors to the hysterical anæsthesias, a characteristic which is naturally shared by the psychoneuroses of war.

As they are not obvious to the patient who presents them, but are discovered in the course of examination, the anæsthesias or analgesias of soldiers are not

in themselves a cause of detention in hospital or discharge from military service. They are never a predominant or isolated sign, but are part of a symptomatic picture of functional order : hemiplegia, convulsive crises, tremor, paralysis, etc., during the course of which they may be discovered if they are looked for. The patients do not direct attention to them during the first examination.

This is why we have commenced by saying that the anæsthesias, from a practical standpoint, only occupy a minor position in the neurology of the war.

However, the importance attributed in classical literature to objective disorders of sensation in the neuroses, and especially in hysteria—a view still upheld by most medical men—compels us to discuss the question in some detail.

But to begin with, it is absolutely necessary to decide what diagnostic value should be given to the so-called hysterical anæsthesias and in what sense they should be considered.

We have adopted Babinski's method of testing sensation in all the cases that have come under our observation (Roussy and Boisseau).

He gives the following directions :—The patient, with closed or bandaged eyes, should not be asked to say whether he feels distinctly or less distinctly a touch, prick, or warmth at some point of the body under examination ; he should not be asked to say " yes " or make some sign at each touch or prick, as this will lead to his attention being drawn to the examination. He should be told to point at once with his finger to the site of the prick or touch.

By proceeding along these lines, we have never yet discovered anæsthesia or analgesia in patients who have been subjected to bombardment or in any of the psychoneuroses that had not previously undergone medical examination. Further, we have often failed to find anæsthesia described in the patient's report as having existed some days or weeks

previously. Use has often to be made of counter-suggestion and, while testing the sensibility, of reprimanding any patient who hesitates to make the required indications. Very satisfactory results are thus obtained, as satisfactory as in the regions reported sound.

Thus we agree with Babinski that the hysterical or pithiatic anæsthesias, in common with the analgesias and hyperæsthesias, are the result of medical suggestion or any other form of suggestion to which the patient has been exposed. They have a certain value in differential diagnosis, as they are the stigma of pronounced suggestibility.

The frequency of the anæsthesias of war is great, judging from the remarks and facts noted in the case-sheets and tickets attached to patients sent into hospital. This frequency is the result of the methods of examination employed. For as it is the current practice to make a systematic test of sensation in nervous cases in all stations from the first-aid post to the general hospitals, as the patient passes through the field-ambulance, the field-hospital, and the casualty clearing-station, etc., it may safely be said that almost all such patients present at some period objective disorders of sensation.

The time and mode of onset of these anæsthesias are impossible to specify. Not appreciated by the patient himself, they remain unknown to him until discovered in the course of an examination. It only exceptionally happens that the patient himself draws attention to the anæsthesia, which should then be regarded with suspicion as suggesting a previously hysterical subject with a recrudescence of his former symptoms.

The clinical characteristics are those described in every text-book and are so well known as to need no detailed description here. Analgesias rather than anæsthesias, of variable extent from small patches

to large areas, they may be complete, affecting all varieties of superficial sensation (touch, pain, heat and cold) and of deep sensation (sense of position, stereognostic sense). They are usually more profound than organic anæsthesias, have a distribution variously termed segmentary, glove and stocking, etc., and do not correspond to the area supplied by a peripheral nerve or nerve-root. They may, however, occasionally be found in the limbs with a distribution in parallel bands roughly resembling a radicular distribution; this is because a search is made for disorders of sensation along these lines.

Lastly, and this is an important point that must be strongly emphasized, the hysterical anæsthesia is no inconvenience to the patient. If not suffering from any motor disorder, he is easily able, with closed eyes, to execute small movements for which control over superficial sensation is indispensable, such as buttoning or unbuttoning the coat, picking up from the table a small object, such as a pin, etc.

Anæsthesia or analgesia, hyperæsthesia or hyperalgesia are seldom isolated symptoms. They nearly always form part of a more complex syndrome in which motor disorders such as hemiplegia, paraplegia, or monoplegia are the predominating phenomena.

The duration of these affections, like that of the other psychoneuroses, depends on their early diagnosis and treatment. They all rapidly disappear on treatment by the faradic current, often in a single séance. Being much less deeply-rooted than the motor disorders, they are the first to disappear when combined with the latter.

Differential Diagnosis.—A psychical anæsthesia or analgesia is easy to recognize on account of its special characters, its distribution and the symptoms which accompany it—paralysis or contracture of a pithiatic type.

This diagnosis is somewhat more difficult in

mixed hystero-organic cases—musculo-spiral or facial paralysis with added disorders of sensation—but even here a little thought will show the real nature of these anæsthesias.

In our opinion a large percentage of objective disorders of sensation of a paradoxical nature, found in soldiers with recent or old-standing wounds, belong to this group and are often only the result of suggestion at the hands of the physician.

In the more complex cases, difficulties in differential diagnosis may arise. For example, as a result of a cranial wound over the motor region of the brain, a patient may develop a hemiplegia or a monoplegia on the opposite side, with anæsthesia. Such a case may be put down as of organic origin; one may think of a hemiplegia with hemi-anæsthesia either as of thalamic origin or as a sensory syndrome of cortical origin (Dejerine's sensory cortical syndrome). But in such cases the anæsthesia affects both deep and superficial sensations, sometimes exhibiting a radicular distribution (radicular cortical distribution) and lastly, in cases of thalamic lesions, it is accompanied by pain, hemi-chorea, or hemi-tremor.

On the other hand, the results of treatment will confirm the diagnosis of a functional disorder, since the anæsthesia rapidly disappears under the influence of the faradic current.

To sum up, it is clear from what we have said that although these so-called hysterical anæsthesias may present some points of interest in the neurology of the war as signalizing a psychopathic condition, their diagnostic value has been grossly exaggerated. If the physician would refrain from a systematic examination of these sensory disorders, of secondary or negligible importance, or if he adopted a more rational method of conducting the tests, the frequency of these hysterical anæsthesias would be, to say the least, very sensibly diminished.

Chapter V

PSYCHICAL DISORDERS OF THE SPECIAL SENSES

Ever since the commencement of the war, sensory disorder of psychoneurotic type have played such an important part that they, above all others, have been witness to the effects of the powerful modern explosives on the nervous system.

Disturbances of hearing are the most prominent of these both in importance and frequency. They appear isolated—deafness, or associated with disorders of the speech—deaf-mutism.

Visual disorders are of rarer occurrence ; disorders of the senses of smell and taste are only exceptionally observed.

§ 1. DISORDERS OF HEARING AND SPEECH

We shall describe together the disorders of hearing and speech, as they are often combined to form part of the same clinical picture.

They form a unique clinical type, almost unknown before the war, even after some disaster such as fire, shipwreck, or earthquake, and it may therefore be said that they form a pathological entity created by the present war.

The **Causal conditions** of auditory disorders are almost always violent bombardment, explosion of a mine, aerial torpedo, or large shell close at hand or at some distance, without producing any apparent external wound. They form part of the concussion syndrome and are one of the most constant elements

in it. It is only exceptionally that they are of spontaneous origin or occur in a patient with an actual cranial injury, fracture or wound in a limb.

The **Onset** is sudden and occurs after a loss of consciousness of some hours or even days, occasionally without previous loss of consciousness; the man comes to himself in a dazed condition, chatters to himself like a madman, raises himself or is carried to the first-aid post by stretcher-bearers, and on attempting to reply to the officer who questions him, finds that he cannot hear and is unable to speak. All information of the course of events with regard to the patient is given by the stretcher-bearers or the orderlies. The patient himself, once recovered, remembers the explosion and nothing more until the moment when he comes to himself in the field-ambulance.

We will give, as an example, an account written by one of our own patients:

On the 16th January I left for Verdun. In front of the village of . . . Then I was present at the capture of trenches on Hill 304 and then . . . During the day, we were under bombardment with guns of large calibre by the enemy artillery. I escaped with contusions. In the evening, when the bombardment had ceased, I got permission from the adjutant and the lieutenant to go to the first-aid post. Here I was massaged by the major. In returning to the trenches, I had with difficulty covered some 500 metres when a large shell fell two metres away. At this moment I lost my memory and started to run, whither I knew not. When a stretcher-bearer met me, he asked me where I was going. I made him understand that I could not speak. Then he took me to the first-aid post.

I remember the major questioning me. I made a sign that I was unable to speak. Then I do not exactly remember what happened. I know that from the field-hospital at Vittel I was taken to the Cérès hospital, where I remained eleven days without being able to

speak ; from there I was transferred by the major to the hospital at Scey-sur-Saône.

This patient was cured by application of the faradic current on the day after admission.

According to their **clinical forms,** disorders of the sense of hearing present various aspects :

(1) **Deaf-Mutism,** the most frequent type, seen at the first-aid post, field-ambulance, or in hospital in three different forms (Roussy and Boisseau).

A. It may occur in soldiers in association with mental confusion ; the expression shows terror, the eyes are haggard, respiration panting and appearance anxious. The soldier is agitated and frightened, wishing to fly from the world about him and hide himself. Immediate use of therapeutic measures sometimes secures the return of speech and hearing, but in view of the restlessness, it is better to wait a few hours or days before attempting treatment. This is the type of deaf-mute with *delirious mental confusion.*

B. Sometimes the patient exhibits listless mental confusion with an expressionless, immobile face, oblivious of everything surrounding him. No sudden noise or any mode of treatment will rouse him from his condition of torpor. This is the type of deaf-mute with *dull mental confusion.*

In these two types the deaf-mutism is part of a train of symptoms of psychopathic order. It is a question of mental disorder of concussion or emotional origin accompanied by deaf-mutism. There is often insomnia, nervous agitation at night, and sometimes refusal of food ; often, during the first few days, there is a slight rise of temperature and occasionally attacks of night-terror.

C. In a third type the clinical picture is different ; the patient is perfectly conscious and seeks to draw attention to his deaf-mutism. He makes numerous gestures, points to his lips and makes signs that he wants to write ; when given the materials he writes a very detailed account of the accident. These are

the deaf-mutes described by Roussy and Boisseau as *loquacious by gesture*, and they resemble the classical type of hysterical deaf-mute.

(2) **Deafness alone**, unilateral or bilateral, is accompanied by subjective auditory symptoms—ringing sounds, noise of water and buzzing, the last being especially common. This type without alteration or modification of speech is rather less frequently seen than deaf-mutism. The deafness may be more or less complete or only be brought out on speaking very softly and is exaggerated by any lack of attention on the part of the patient.

In any case, it is identified by an important clinical characteristic which serves to differentiate it from organic deafness. The patients are little inconvenienced by their deafness ; *everything occurs as if it only existed at times when the patient's attention is directed to it by investigation.* We have often come across patients suffering from concussion deafness, examined for the first time a few hours after the shock, and told them sharply to shut their eyes ; taken by surprise, they almost always obey the order ; subsequent attempts do not meet with success. Further, psycho-neuropathic deafness, in distinction to organic lesions of the ear, does not produce changes in the timbre or elevation of the voice ; this is an important sign which, as we shall see, can be made use of in differential diagnosis.

(3) **Mutism** may also exist alone, without deafness. It is absolute and total ; the patient emits no sound, makes no movement of the lips and shows by many gestures that he is incapable of speaking. The tongue is immobile and cannot be protruded or moved voluntarily. Naturally enough, automatic movements, *e.g.* of mastication, are preserved. In the cases of dumbness or deafness existing alone, as in the more complex conditions where these are combined, various changes in the psychical

state may be found, and these are described in the section dealing with deaf-mutism.

In some cases the nervous shock is succeeded, not by mutism but by simple **aphonia.** It sometimes exists from the first, but often follows mutism when the latter is on its way to recovery.

(4) **Stuttering** may exist alone or be associated with other symptoms of shock ; it appears under the same conditions as the preceding disorders, with which it may be combined ; for this reason and for convenience of description it is included in this chapter. It resembles the classical type of hysterical stutter ; its symptoms are always very pronounced, with a sudden onset and rapid or immediate cessation under the influence of electrical treatment.

This hysterical stuttering must be distinguished from true stammering, which can easily be done by the history and also, if opportunity occurs, by therapeutic methods. But it must be borne in mind that in true stammerers the multiple emotions and shocks of war may suddenly increase the difficulty of phonation and give it the appearance of mutism or hysterical stammering. A few days' rest under observation generally suffice to mitigate the disorder and clear up the diagnosis.

There are other disorders of speech, such as " nigger-boy " speech, which may for a moment be mistaken for aphasia (motor pseudo-aphasia).

Associated Signs and Symptoms.—As regards the larynx, examination shows that the vocal cords are in the position of rest.

In the ears, perforations of the tympanic membrane, sometimes accompanied by otorrhœa, may be found —organic lesions on which the psychical nervous disorder has been grafted. These more severe lesions are rare ; more often slight lesions, chronic and long-standing, are found, such as adhesive otosclerosis, scars, etc., suddenly aggravated by the shock.

Lastly, one often finds labyrinthine lesions, with vertigo, buzzing in the ear, disorders of equilibration and decreased acuteness of hearing, which appear more clearly when the neuropathic symptoms have passed away.

Lumbar puncture occasionally reveals increase of albumin with slightly increased pressure and lymphocytosis, but this is uncommon. In the large majority of cases the cerebro-spinal fluid is normal.

Headache is common ; it may persist for several weeks after the disturbance. Rise of temperature also often occurs in the severer forms, but usually abates after a rest of a week or fortnight.

The Course of these disorders of hearing of psychoneurotic type, with or without added affection of the speech, depends directly on the therapeutic measures employed and the time of their application.

Though in old cases of deaf-mutism due to shock it is difficult to effect a nervous stimulus necessary to bring back the functions of hearing and phonation, it is, on the contrary, extremely easy to " cure " patients whose affection is of recent date.

Recovery is either *immediate* (obtained at once) or *rapid* ; only exceptionally is it *slow*, that is, twenty-four to forty-eight hours after the onset.

When the patient comes round, he speaks in monosyllables, " nigger-boy " jargon, or in a variety of ridiculous ways ; in other cases there is aphonia. It is only necessary to continue or increase the electric current for a little while to bring about complete and lasting recovery. Thus there is no need whatever to resort to the long and involved methods of auditory re-education or to lip-reading, still far too often practised by specialists. Such procedures are not only useless, but they have the effect of directing the attention of the patient to the disease from which he believes himself to be suffering and thus of compromising or retarding recovery.

Relapses may occur, as in other psychoneuroses.

They are commoner in the more severe forms—sensory disorders with a confusional condition—and come on when the patient is sent back too soon to the firing-line and is again subjected to a violent bombardment.

With regard to the **Nature** of these disorders, it is quite evident that we have to deal with a psychoneurosis of emotional or concussion origin, resulting from emotional shock. They are quite on a parallel with the tremors or impotence of the limbs, the loss or disorder of speech being one of the modes of reflex outward manifestation of the emotion. As for auditory affections, it is easy to understand that the intense bombardments and the violence of modern explosives may cause auditory disorders, deafness etc., a matter of common knowledge to all who have been under heavy fire ; such factors are well calculated to produce neuropathic manifestations in predisposed individuals with an already unstable nervous system.

The fact that at the start it is a genuine and real neurotic disorder cannot be doubted. It is also equally certain that patients who have unfortunately not been treated in time develop the phenomena of fixation or exaggeration more or less subconsciously. The question of routine practice in such cases is the same as arises in other psychoneuroses. We have discussed them in the chapter dealing with malingering.

The Differential Diagnosis of sensory auditory disorders must be considered as one of great ease, if the correct importance is attached to the special conditions of their onset and to their stereotyped and almost pathognomonic clinical characteristics—sudden onset following nervous shock, a manifestation of emotional shock as one symptom of the concussion syndrome, the various clinical aspects

of their symptoms with the common characteristic of rapid recovery under the application of psychotherapeutic methods.

One only needs to have seen a certain number of such cases to recognize them at once and with certainty.

Cases of deafness merit some further consideration. Judging by the observations published by specialists, it is evident that the question of *deafness of war* is a complex one, and opinion is still very divided.

Castex divides the war injuries of the auditory apparatus into two chief groups :

(1) Rupture of the tympanic membrane ; (2) labyrinthine disturbances.

Rupture of the tympanum is usually produced when a projectile (bullet or shrapnel) strikes the temporal or mastoid region or when a shell explodes without great intensity.

Labyrinthine disturbance is observed when a large-calibre shell blows up a trench and throws its occupants into the air. It is often part of a cerebral concussion. The prognosis of future hearing is very grave. The deafness tends to increase as the scar-tissue invades the delicate structures of the internal ear.

These cases of war deafness due to labyrinthine disturbances are much more severe than those occurring in workmen's accidents. The causal conditions are so much more intense.

According to Lannois and F. Chavanne, the prognosis of cases of deafness depends :

(*a*) On the previous condition of the auditory apparatus. Patients suffering from chronic otitis media or already pronounced otosclerosis form a far larger section of cases of deafness or impaired hearing after labyrinthine concussion due to a bursting shell than do individuals previously in good health.

(*b*) On the presence or absence of direct cranial trauma. Deafness is the rule in traumatic mas-

toiditis, common in extensive facial trauma in the neighbourhood of the ear, rare in fractures of the cranial vault. Deafness is therefore usually unilateral.

These authorities state that in the absence of direct trauma, labyrinthine disturbances rarely lead to total deafness; of 615 cases of labyrinthine disturbance without rupture of the tympanum, they only observed permanent bilateral deafness in 2 per cent. When it occurs, however, it is extremely serious; acoustic remedies have no influence on it; recourse must be had to lessons in lip-reading in order to make social life bearable to these unfortunate patients.

Without wishing to exceed our proper scope, we must take this opportunity, based on our own personal experience, of drawing attention to the frequency of false labyrinthine disturbance created solely by medical suggestion and to association of hysterical with organic phenomena.. Otologists, perhaps, do not always ascribe to neuropathic phenomena in the wounded their merited place, and do not adequately provide against the risk of influencing the patient by suggestion when making their examination.

Of the various methods recommended to distinguish inorganic from true deafness, the "deafening method" of Lombard renders excellent service. It is performed as follows:

A normal subject raises the spoken voice when both ears are sufficiently muffled by any suitable means. This phenomenon appears to result from sudden suppression of auditory control over the intensity of the sounds emitted during normal phonation.

This deafness may be simply procured by means of telephone receivers placed to the ears and connected up with a dry battery and the circuit interrupted by a simple continuous ringing apparatus.

Instead of by telephones, temporary deafness can be produced by passing a current of water through the two auditory canals or by insufflation of air at the external meatus on both sides by means of an insufflator and a double indiarubber tube.

Psychoneurotic *mutism* is so characteristic as to preclude any error of diagnosis.

Lastly, *aphonia* occurring suddenly as a result of emotional shock is not likely to be mistaken for an organic lesion of the larynx. Interrogation and examination of the larynx will reveal organic lesions of the vocal cords existing prior to the trauma and producing dysphonia or aphonia.

§ 2. VISUAL DISORDERS

Neuropathic affections of sight are much rarer than those of hearing. Perhaps this is because these patients are more quickly sent on to the special departments and thus have a better chance of avoiding the danger of medical suggestion. Perhaps also because the auditory mechanism, much more than the visual apparatus, is submitted to so many disturbances (shock, bombardment) which establish and fix the neuropathic phenomena.

Also it must be remembered that ophthalmologists have at their disposal means of examination much more perfect and certain than that of otologists. There is no doubt that this factor reacts on the prophylaxis of ocular neuroses.

Although rare, they are by no means of exceptional occurrence.

They appear after a shell has burst at a more or less distant point, sometimes after simple atmospheric disturbance, but usually after earth or dust has been thrown up into the eyes, sometimes as a

result of asphyxiating shells or gas or tear-shells. In some cases the symptoms follow the simple presence of some foreign body in the eye or some slight trauma without any previous shock.

From a *clinical* point of view, disorders of vision are sometimes isolated and constitute the only symptom for which the patient is sent to hospital; in other cases, they are associated with further neuropathic manifestations, such as tremor, disorders of the gait, affections of the hearing, convulsive crises, etc.

Both visual disorders and motor affections of the ocular apparatus and its appendages are observed. Complete **Amaurosis** is not rare. These supposedly sightless patients have been sometimes for several months in various neurological departments, in whom examination of the eye has revealed the hysterical nature of the blindness.

Amblyopia and **Photophobia** are frequent phenomena; they usually accompany other neuropathic disorders such as hemiplegia or paraplegia. They are patients who wear large smoked glasses, who complain that their sight has been affected since the explosion of a shell or the discharge of gas, who complain of indefinite tingling and watering of the eyes, and who have been recommended to wear dark glasses. All these phenomena yield to psychotherapy.

Blepharospasm (uni- or bilateral), **Movements of the Lids** or **Blinking** of neuropathic type are by no means rare. **Nystagmus** is more exceptional, a few cases only having been recorded.

We will not go into details regarding the *course* of these visual disturbances, as the same considerations hold as in auditory disorders.

When unrecognized as such, they may persist for a long time, even indefinitely; diagnosed and treated in the approved manner, they are certain of immediate recovery.

To complete this chapter, we must just mention the rare conditions of neuropathic **loss of smell** and **loss of taste** following a shock or trauma of war. They are of exceptional occurrence and as neuroses of war offer no special peculiarities ; like the objective disorders of sensation, they are usually the result of medical suggestion. There is nothing easier, indeed, than to create a hemi-ageusia or hemi-anosmia in a highly neuropathic individual. But it is just as easy to effect its disappearance.

Chapter VI

DISORDERS OF SPHINCTER CONTROL

The existence of affections of the sphincters in the psychoneuroses—notably in hysteria—is still recognized in standard text-books. On the other hand, Babinski and many other neurologists deny that hysteria alone can create permanent and lasting disturbances of function in vital organs and ascribe any visceral affections to intentional deceit or to some unrecognized organic affection.

These new views have by no means yet pervaded the medical profession, which, with its naturally conservative point of view, tends to remain faithful to the attractive doctrines (not to say dogmas) of the Salpêtrière school.

As will be realized from this short preface, we are not devoting this chapter to sphincteric disorders in the psychoneuroses of war because we accept the old-established pathological limits of hysteria, but because the present circumstances, so favourable for medical observation, compel us to cite the facts we have noted during a period which has abounded in neuropathic affections. We may say in advance that these facts throw their weight on the side of Babinski's theory.

§ 1. Disorders of Micturition

Judging from the diagnoses found on case-sheets and discharge-papers, disturbances of the vesical function are of frequent occurrence. Frequently enough, soldiers are sent from their regiments to

the Army Neurological Centres owing to neuropathic vesical disorders; just as numerous are the cases which, in the interior, are transferred from the medical, surgical, or even urological departments to the special institutions for nervous diseases.

INCONTINENCE AND RETENTION OF URINE are the two most frequent disorders, and are the only sphincteric affections we need consider.

A. **"Hysterical" incontinence of urine,** or better, **false incontinence,** in soldiers is more common than retention, and for a very good reason! Not accompanied by any painful symptom and not disturbing in any way the function of the organ, it nevertheless always necessitates the rapid discharge of the patient, who wets his clothes by night and day, wets the mattress on which he sleeps and becomes an unpleasant companion to the men in his company. "This patient is an annoyance to his comrades and cannot be allowed to remain in the corps" is the usual remark which figures on his discharge-paper.

Cases of psycho-neuropathic incontinence of urine observed during the war fall into four different groups:

(1) *False incontinence due to shock.*—In this first type the patient, after some violent bombardment or temporary burial, presents the various symptoms of the concussion syndrome—loss of consciousness, tremor, deaf-mutism, paralytic motor disorders, etc., to which is added incontinence of urine. The patient, at the moment of the shock, has passed water, a phenomenon easily explained by the emotional reaction. Little by little this disorder becomes chronic and fixed subconsciously; this fixation is especially furthered by erroneous diagnosis or injudicious medical examination; thus the involuntary escape of urine becomes definitely established. In such patients, tests of the tendino-muscular and cutaneous reflexes and perineo-scrotal sensation exclude any organic lesion of the cord or cauda

equina. Moreover, the timely application of psychotherapy soon puts an end to this false incontinence.

(2) *False incontinence due to habit.*—In this variety the involuntary escape of urine—usually nocturnal, rarely diurnal—commences without any apparent cause or follows some minor incident such as a slight febrile affection or some vesical derangement. These are soldiers who have long been in various hospital departments, who have gone through their military training without being discharged, and in whom the incontinence of urine has only been noticed since the war. They are sometimes individuals with mental deficiency; such cases were specially common in the army last year when Dalbiez' measure came into force. No organic mischief is revealed on examination. It is not a case of so-called essential incontinence, first because the onset dates back several weeks or months in men of twenty, thirty, and forty years of age; secondly, because energetic re-educative treatment always puts a stop to it. They are men who have got into the bad habit of wetting the bed—*habit-piddlers*—somewhat resembling those who have contracted the habit of limping. We can apply to them all the considerations discussed under claudication; the same pathogenesis is at work in their origin and fixation, and the same remarks apply with regard to the genuineness of the disorders under observation. But it must be understood that here, again, there is a real, irritative organic lesion at the basis of the perversion of function of the organ in question; it is the persistence of the incontinence, or its association with the condition which produces it, which is illogical and paradoxical.

We will only point to the need of carrying out vigorous treatment, instituting a strict inspection of the bed-clothes and garments of the patient, assisting treatment by an application of the faradic current to the perineum and, if necessary, by an intrathecal injection. In a very few days the symptoms, wrongly

considered to be particularly intractable, will be found to disappear.

(3) *Recurrence of "essential" incontinence of urine.*—In other cases there may be attacks of true incontinence after a freedom for some years, provoked by fatigue, emotion, or shock, comparable to epilepsy. These are previously discharged men or reservists who have recently joined up, in whom the history and mental examination reveal evident psychopathic stigmata. These manifestations, always implying some appreciable mental defect, do not directly concern the psychoneuroses of war, but are rather psychopathic disorders of a constitutional type, which the events of war have only rendered more apparent.

Psychotherapy is not applicable to such cases. They are soldiers of no value to the army and are only fit for sedentary work or discharge to class 2.

(4) There remains, lastly, a *type of incontinence often wrongly described as hysterical or psychopathic*, which, though rare, is important to recognize, as it is the object of many incorrect diagnoses. It is an incontinence which follows a period of incomplete retention or a short period of complete retention, and which is due to a slight lesion of the cauda equina or conus medullaris, a lesion which only produces an incomplete clinical syndrome and often passes unobserved unless sought for with care. One of our own patients was for a long time treated and considered as an ordinary incontinent pyschopath (and even as a malingerer), when he was really suffering from an organic lesion of the cauda equina which had not been discovered.

B. **Hysterical retention of urine** is much rarer than incontinence, as will be seen by examining the diagnoses on the case-sheets. We have given the reason above.

In our opinion there is no such thing as true hysterical retention.

In this type, again, we have to deal with patients suffering from concussion, who immediately exhibit retention of urine and are sent back as such.

In all the cases that have come under our observation and which we have been able to examine thoroughly, either at the front or behind the lines, we have found either that—

(1) The retention has only been transitory, lasting some hours or days after the concussion and necessitating the use of a catheter: These are transient spastic phenomena, of concussion or emotional type, and disappear spontaneously, or

(2) The retention has been incomplete, with vesical pain, necessitating the passage of catheters at the field-hospital or ambulance-train. Tests of the vesical reflexes by a genito-urinary surgeon show a normal condition of the bladder. But the neurologist will find increase of the tendon and bone reflexes and sometimes ankle-clonus, hypæsthesia of the perineo-scrotal region, abolition of the anal reflex (uni- or bilateral), or changes in the cremasteric and abdominal reflexes. Lastly, lumbar-puncture may reveal lymphocytosis or slight increase of albumin.

In some cases, then, there will be indefinite signs of a unilateral or bilateral lesion of the cauda equina; in others (where lymphocytosis is found), signs of a lesion of the cord, often of a specific nature. These are abortive organic lesions which it is important to diagnose.

No case of "hysterical" **anuria** has been observed, to our knowledge, during the war, nor is this surprising. Further, the days have long since passed since hysterical **anuria** was recognized as a definite entity, even by partisans of the classical doctrine of hysteria.

Polyuria or **Pollakiuria.**—Though these may be found as transient manifestations or reflex emotional reactions, they are only of short duration unless caused

by a latent organic lesion of the bladder or kidneys, previously unrecognized.

§ 2. Disorders of the Ano-rectal Sphincter

These are only mentioned in order to deny their existence as psychoneuroses. Apart from *emotional diarrhœa,* common on the battle-field, and relaxation of all the sphincters in cases of severe concussion, the cases one sees (and this only exceptionally) are those of false incontinence in the weak and degenerate, who more or less unconsciously soil their clothes with urine and fæces and who may be improved by a severe reprimand.

To sum up: of the cases drawn from the vast field of observation at the present time, it will be seen that none of those studied in this chapter comes within the old meaning of the word "hysteria." They all show other and different characteristics on thorough examination.

Chapter VII

VISCERAL DISORDERS

In the group of physical phenomena produced by emotion we must take into account the physiology and pathology of visceral disturbances. As we know, the emotional states do not find expression solely in modifications of sensation and of motor function, but also in extensive though transient disturbances of function of the viscera. It is a strange phenomenon that psychoneuroses of an emotional nature react at times on the functions of certain viscera, and it is also surprising that the mechanism of these disturbances still remains a matter of pure speculation.

The visceral complaints which may result from emotion are extremely varied, and it may be said that there is no system of the human body in which the emotion may not find expression. But these *immediate* emotional disorders are essentially transient, and when the cause of the emotion is no longer at work they vanish without leaving any trace.

The same cannot be said of certain affections which may, *in a neuropathic individual,* become fixed for a more or less protracted period. We shall discuss digestive, cardiac, and respiratory disorders.

§ 1. Digestive Disorders.—Hysterical or mental anorexia and the disturbances of sensation in the intestinal tract which they present are matters of common knowledge. We shall not discuss them here, as we have not seen a single example during the war.

Vomiting deserves attention, being relatively common. It is contradictory in its behaviour and cannot fail to be recognized when such a case has once been witnessed. After a fairly long spell in the trenches, a young soldier, of previous good health but who has occasionally suffered from digestive trouble at the front, is sent back on account of persistent vomiting.

At the first examination the observer is surprised at the more or less perfect preservation of the general condition of the patient and doubts the reality of the vomiting. Careful watching excludes deliberate deceit, however, and shows the reality of this abnormal gastric phenomena. Occurring several times a day, without apparent cause, this neuropathic vomiting occurs without any great effort and terminates without the patient experiencing any real fatigue. It is essentially alimentary, semi-digested food being found in the vomit; sometimes there is slight regurgitation of bile.

Examination fails to reveal any abnormality of the stomach. It is not hypersensitive, and splashing is not heard or felt. The liver is normal.

If, as in our own cases, an X-ray examination is made after a bismuth-meal, one finds either that the stomach is absolutely normal and is emptied in a normal length of time, or that, while retaining a normal motility, the pyloric canal lies somewhat lower than normal. This slight gastroptosis can hardly be held to explain the origin of the vomiting.

One of the most striking peculiarities of neuropathic vomiting is that it by no means impairs nutrition. The patient preserves his appetite and loses little weight, a certain proof that in spite of its frequent bouts, the vomiting nevertheless allows a fairly large quantity of chyme to pass into the intestine.

The *diagnosis* of neuropathic vomiting, as we see,

offers no difficulty. It must not be made, however, until all organic causes have been eliminated, especially ulcer in the region of the pylorus.

There is often some underlying organic condition which must be sought for and treated as soon as the neuropathic element has disappeared. There seems to be no tendency to spontaneous cure in this neuropathic manifestation. Certain patients reach us after having been in various medical departments and institutions, where they have, perhaps, been rather neglected owing to the intractable and discouraging tenacity of the vomiting.

Great attention should therefore be paid to these patients and treatment should consist both of a strict dietetic régime from the outset, accompanied by psychotherapy.

With regard to their *nature* and *genesis*, the same considerations apply as those given for incontinence of urine (*cf.* p. 813).

§ 2. Respiratory Disorders. — **Neuropathic Tachypnœa.**—These are much less common than the digestive disorders and much more difficult to distinguish. It is true that there is disturbance of the respiratory function in every emotional condition, taking the form either of acceleration or retardation of respiratory movements; but it is quite exceptional for this affection to become fixed for a sufficient length of time to permit of careful observation or to exhibit recurrences such as we have discussed above.

All regimental medical officers have seen, after violent bombardments, soldiers a prey to prolonged emotion. In these cases the rapid respiratory movements are the striking feature; when at rest in a place of safety, stimulated by cheery encouragement, the patients quickly become quiet and the rapid breathing disappears.

This tachypnœa may, however, recur when the patient has been brought to the field-ambulance

or even in a base-hospital. This is *neuropathic paroxysmal tachypnœa,* of which there have been a number of cases during the war.

There is a great point of distinction between this purely functional disorder of emotional origin and affections of similar character, but due to visceral lesions ; it is that this tachypnœa is always accompanied by blatant, excessive and disproportionate manifestations. The disorder is not limited to an acceleration of the respiratory movements, but the patient is seized by general agitation. This is often a striking feature during an attack of tachypnœa : the patient groans or excitedly shouts for help, his limbs are shaken by disorderly movements or by a tremor. The rate of the respiratory movements may be considerably increased, but it is often really lower than it appears. The excursions of the chest-wall are wide, expiration and inspiration are noisy and interrupted by guttural sounds, appeals for help, or groans.

In spite of this impressive respiratory condition, the pulse remains quiet, hardly increased at all in frequency.

This so-called polypnœa of exertion is increased not only by the slightest movement, but even by such trivial actions as replying to questions or shaking hands, and this when the patient is lying comfortably in bed. The mere fact of someone entering the room suddenly is enough to start a crisis.

A severe reprimand and a few blows on the chest from a towel wrung out of cold water often suffice to cut short an attack.

The same may be said of isolation ; as long as the patient is not watched by a friend, the doctor, or a nurse, he has no attack of tachypnœa. As in all the extravagant manifestations of the psycho-neuroses, they need for their production and development a sphere of interested spectators.

We must add that true neuropathic tachypnœa is rarely a single, isolated phenomenon. A careful

examination will usually reveal other manifestations of the neurosis.

§ 3. Circulatory Disorders. — **Tachycardia, Bradycardia.**—Like the last group, these also form an integral part of the emotional states. They are much more likely to recur than are respiratory phenomena. Cases of psycho-neuropathic tachycardia are not uncommon even in the general hospitals in the interior.

Psycho-neuropathic tachycardia, like tachypnœa, is characterized by its occurrence in *paroxysmal attacks, which are brought on by the revival of an emotion.*

The cardiac disturbance is not usually the only feature, the patient at the same time exhibiting other psycho-neuropathic manifestations—paralysis or contracture, disorders of the gait, stuttering, etc.

The attack of tachycardia is brought on by the remembrance of a previous emotional state. This recollection may be brought about by medical inquiry—a common occurrence, though its connection with the recrudescence of the emotion is not quite clear; or by some perception which is somehow connected with the idea of the battle.

Unlike true paroxysmal tachycardia, the attack does not come on with the same suddenness and is not accompanied by any initial cardiac shock nor by any distress. The pulse-rate rapidly rises and the arteries pulsate visibly under the skin, but the patient is not specially embarrassed. It may reach 120-140 per minute and vary round this figure for several hours. Then the heart quietens down and the rate becomes normal.

The attack is often of much shorter duration, lasting not more than half an hour; in exceptional cases the tachycardia may persist for several days, sometimes appearing and disappearing without apparent cause.

Claude and Lhermitte have observed cases of

prolonged attacks of tachycardia alternating with hysterical convulsive crises. In these patients there was no cardiac lesion whatever and no alteration in the vascular tone or arterial tension; the renal functions were normal.

Although we by no means intend to refer these hysterical convulsive crises and tachycardia to the same origin, it is interesting to note the occurrence in the same patient of two manifestations, the sources of which are fairly closely related—the extravagant motor agitation of the convulsive crisis and tachycardia.

Psycho-neuropathic tachycardia never produces very alarming cardiac disorders and never causes cardiac failure.

More rarely, the psycho-neuropathic condition takes the form of a slowing of the pulse-rate. In two cases observed by Claude and Lhermitte, *paroxysmal attacks of bradycardia* alternated with severe fits of convulsive hysteria. In spite of the obscurity which still surrounds the pathogenesis of such cases, we have thought them interesting enough to mention in passing.

Tachycardia and bradycardia are particularly obstinate to any form of treatment.

Chapter VIII

NERVOUS ATTACKS

(*Les crises nerveuses*)

The nervous crises of psycho-neuropathic origin form, we believe, a natural stepping-stone between the disorders we have just described and the purely psychical manifestations. Although these psychical disturbances may not be of premier importance amongst the various " crises " that may be observed and are difficult to analyse, yet they form an integral part of the clinical picture and in some cases are of the greatest importance. Their motor and sensory phenomena are well marked and at once distinguish such attacks.

§ 1. THE FIT OF TERROR

(*La crise d'anxiété*)

We need give no description of paroxysmal attacks of terror and mental anguish ; they are well known, and from the point of view of the war present few peculiarities. It must be clearly understood that these outbursts of terror or anguish may occur in even the most courageous soldiers ; further, that these manifestations do not represent an intense and extravagant reaction to the legitimate emotion of the battle, as we might be tempted to imagine, but simply some previous disorder which the war has not sensibly modified.

On the other hand, symptoms of extreme terror observed amongst soldiers in the firing-line itself are much more frequent and characteristic. During

a preparatory bombardment or an artillery barrage by the enemy, men may be found incapable of any gesture, terror-stricken, fear written upon their faces; these men are quite unable to reply to questions put to them. Respiration is rapid and irregular, the heart-rate much increased. Some, in an effort to escape from this mental torture, exhaust themselves by various gestures and fruitless agitation; then quite suddenly the terror abates and the soldier becomes quiet and almost serene and placid.

It must not be thought, however, that these essentially emotional phenomena of active service are only witnessed during action; they are almost as common during preparation for an attack. They are seen even at the time of mobilization. Fatigue, overwork and intemperate habits are certainly not unconnected with their production.

These crises of terror and mental anguish must not be confused with common cowardice. The real coward, even at the most critical moments, preserves his instinct of self-preservation; the other, on the contrary, attempts neither to protect himself nor to escape, his actions being inhibited by the very mental torture which he suffers. Lastly, cowardice is practically constant—a coward he is and a coward he remains at every critical moment. The other does not always react in the same way when submitted to the same dangers; often brave and courageous under dramatic circumstances, some relatively slight emotion may precipitate a crisis.

As a rule the fits of terror only occur at long intervals and do not entail the discharge of the soldier. In some cases, however, they develop an intensity and frequency such that the patient becomes incapable of regular military duties and has to be sent back. Under these circumstances the fits of mental anguish are accompanied by somewhat different manifestations. Other and more striking features are added to the phenomena of simple terror, which render the symptomatic picture more com-

plex. Further, the occurrence of a crisis is not determined by an emotion at the time, but only by a recollection of previous emotions which the patient brings to mind.

The memory of these emotions may remain latent and only come to light on the occurrence of some incident with which they are in some way closely associated; but most often the production of this anxiety emotion is stirred up and revived very easily. When one speaks to the patient about the bombardments he has experienced or the attacks he has helped to repulse, his expression soon bears witness to his deep emotion. He rises excitedly and becomes agitated, his respiration is accelerated and his pulse quickens. Sometimes the very fear expressed by his eyes seems to beg for respite from the discussion of such an alarming subject. In such a patient, the expression of terror is profound; his whole body shakes, his legs give way, his voice is broken and speech is disturbed. He tries to escape, cowers in a corner of the room or appears to repulse an imaginary enemy. It is quite certain, however, that there is no real hallucination.

In the intervals between the attacks, the patient is quiet and peaceful, though somewhat melancholy and preoccupied. Sleep is irregular, interrupted by nightmares or dreams of war which, by bringing such recollections into consciousness, may engender a crisis of terror.

Although it is not always easy to determine the exact part played by alcoholism in these conditions, it seems that these states of revival of emotion cannot be entirely put down to this factor. Though it is possible or even probable that it plays some part in certain patients, it is undeniable that it is not the only factor. Alcohol may prepare the soil and, so to speak, make the bed for the neurosis; but the essential element is emotion, or more often the sum-total of emotions which, acting on a constitution with congenital or acquired predisposition,

creates this special disposition to revival of the terror.

The *prognosis* of these conditions is, in the great majority of cases, a favourable one and, apart from the emotional features we have described, there is no other disorder of the psychical functions.

§ 2. The Fit of Motor Agitation (*La crise hystérique*)

The severe attack of hysteria major described so minutely by Charcot and P. Richer had almost disappeared from medical observation since hysteria has been treated by strict isolation, until its revival during the war with all its wild and extravagant characteristics. In the trenches, at the field dressing-stations, in the field-hospitals and in the interior, there is not a medical officer who has not had occasion to observe numerous cases of "nervous crises" of a neuropathic or hysterical nature. Unfortunately these are still far too often deliberately labelled "epileptiform attacks" and confused with those of true epilepsy. We shall return to the differential diagnosis later.

Although the cases that have come under our observation do not entirely correspond to the description given by the Salpêtriere school, all the essential elements may, however, be exhibited: tonic and clonic phases, contortionate movements and sometimes periods of "attitude" indicative of the various passions.

Just as in the other psycho-neuropathic disorders we have successively described, each patient modifies the attack in his own fashion and according to his individual personality. In spite of this, the same chain of events is usually observed.

The onset of the attack is striking; the patient makes various contortions, rolls on the bed or throws himself onto the ground. The body stiffens,

the head is thrown back, the eyes are prominent and rolled upwards under the quivering lids. The face is cyanosed, the veins of the neck are distended and respiration is suspended for a short time. Then commences a series of uncontrolled and extraordinary attitudes. The limbs are thrown wildly about, striking the wall or the floor; the hands clutch at the garments or bedclothes, tear or snatch at anything within reach; or the patient may fall to the floor making the most alarming movements, shouting, struggling, throwing himself at the nurses or breaking the windows. Notwithstanding this terrifying condition, well calculated to frighten those who are not familiar with this tumultuous psychopathic agitation, the patient can be restrained fairly easily, and the free use of cold water applied to the face and thorax or the application of a faradic current will soon put a stop to this uncontrolled outburst.

The exciting *cause* of these attacks varies; imitation and suggestion are often at work.

Emotion may be the immediate cause or it may be only the recollection of an emotion that brings on the attack.

When the active, struggling movements have ceased, the patient comes to himself, rather hazy and fatigued, but usually knowing what has occurred.

We must repeat that it is only necessary to treat these convulsive manifestations by energetic psychotherapeutic methods to see them quickly disappear. It is indispensable, too, that "prophylactic surroundings" shall be obtained in any branch of the service dealing with nervous cases; and even the patients themselves know the medical officers whose cases do not have nervous crises.

Do we infer that these crises are pure shamming, under the complete control of the patient and produced with the object of impressing those around him? This would be going too far and ignoring

the rôle of the subconscious which, once the attack has commenced, is at work in the sense of directing the various motor manifestations. But it appears quite evident, and this is based on numerous personal observations, that the patient can, *if he wishes*, always prevent the onset of an attack, especially if helped by the means indicated above.

How many times have we received patients sent back on account of daily and almost continuous nervous attacks, patients who have not exhibited a single relapse in hospital during a period of observation of over a month! A severe admonition on admission and a promise given by the patient have brought about a cure.

It may, therefore, be said that although these attacks are a form of psycho-neuropathic disorder frequently written up on the patients' admission-sheets, they are of exceptional occurrence in a well-organized and properly-managed institution.

Diagnosis.—We cannot discuss here the points of differential diagnosis between hysterical crises and epilepsy.

We will only suggest that in the large majority of cases, the diagnosis is easy. An epileptic fit is distinguished by an initial cry, pallor of the face, mydriasis, biting of the tongue, ecchymoses in the palpebral conjunctiva, spontaneous passage of urine and deep sleep; also by its onset in infancy or childhood and occurrence by day or night without obvious cause. All these features are wanting in hysterical conditions.

It must be admitted, however, that the differential characteristics are not always so clearly defined, and the diagnosis of the nature of a convulsive fit is not an easy matter, even when the physician is able to observe the attack.

Masked forms of epilepsy with an increase in the frequency of the fits are far from rare amongst soldiers.

and may, by their resemblance to hysteria, give rise to difficulty in diagnosis. They must be kept under observation for some time before a definite diagnosis is made and discharge is decided upon. One too often sees the name "hystero-epilepsy" applied to these cases, a term which is only used as a cloak of ignorance.

Chapter IX

PSYCHICAL DISORDERS

In the foregoing chapters we have studied the various motor, sensory and visceral manifestations of the psychoneuroses. We have dwelt upon the fundamental fact that these phenomena, so diverse in their clinical features, have their origin in a psychical disturbance based on the emotions, a disturbance which is common to all these affections and which allows their classification into a distinct nosological group—that of the psychoneuroses.

We must now commence to study those manifestations which are practically limited to the mind, not the psychoses proper but the psychical disorders which fall into the general group of psychoneuroses of war.

The psychoneuroses, with psychical features predominant, are less common than the other varieties, but have been observed all through the war and it must be anticipated that they will by no means completely disappear on the cessation of hostilities. From the standpoint of time of occurrence they might be grouped into three classes corresponding to the three chief phases of war service, *i.e.* psychopathic disorders of mobilization, battle, and rest. This classification, attractive though it may seem, should not be adopted, for the reason that the psychical disorders of the various stages of war do not differ sufficiently from one another either in their clinical characters or etiology.

The same consideration holds for their classification according to the locality in which they develop. Even if there are distinct mental psychoneuroses of the firing-line and of the base (not to

mention those of prisoners of war), it would be difficult to classify them on such a foundation.

The whole subject of the psychical disorders of war is somewhat confused and uncertain and we are compelled to adopt a strictly clinical basis.

Taking the whole group of individuals who, in the course of the war, have presented or present psychopathic symptoms, it is important in the first place to make a clear distinction between those mental disorders which have only been *aggravated* by the events of war and those which have been definitely *caused* by them.

All who have worked, either near the firing-line or at the base, and who have examined men afflicted with mental disorders, have found that certain patients suffering from *delirium of war* were in reality cases of general paralysis of the insane, of mania, of persecutory insanity, or of melancholia; the state of war had only added colour to their madness. In all such cases, the war has only set a special seal on their disordered mental or emotional condition. The disease itself, whether general paralysis or mania, for example, remains the same as in the days of peace; neither its prognosis nor its course has been modified.

The same is the case with mental defectives who, accustomed to a quiet and largely automatic mode of life, have exhibited fits of insanity under the influence of the war, which has disturbed their mental equilibrium. Here, again, military operations have only given fresh ideas to an undeveloped and weak imagination. How numerous are these inventors of destructive machines, these prophets and mystics! They have always existed, and their insane ideas of to-day are but modified reflections of the very ideas held by the agitators of yesterday.

In addition to this first group, there are certain psychopathic disorders which, though similar in many ways, are of different etiology. These are

the psychical manifestations not directly brought on by the war, but the result of fatigue, strain, and especially alcoholism. Cases of this nature have been and are still numerous. It is evident that the state of war itself is not exclusively responsible, but it cannot be denied that it has an unfavourable influence on a weak or previously damaged mind; and it also tends to lead certain individuals, may be owing to their unsettled life and possible idleness, to give way to their craving for alcohol. In any case, these hardly come within our scope and we have only mentioned them in order to contrast them with psychopathic disorders which are, on the contrary, directly associated with the state of war.

The two great etiological and pathogenic factors at work in producing the psychical phenomena of the psychoneuroses of war may be summed up in two words: *emotion* and *concussion.* It goes without saying that these two elements often exist together and are so intricately bound up one with the other that in certain cases it is impossible to decide their relative rôles. Like the various neuropathic disorders already described, they are not based on any material lesions (with the reservations which this term implies) of the nerve centres, and they recover without leaving any trace.

As we have pointed out above, emotion depresses or stimulates, excites or paralyses; and just as in the motor sphere, where a morbid emotion may make itself visible by paralysis or convulsions, so in the mental sphere it may come to light as an inhibition or diminution of psychical activity, or as an unbridling of the immoderate and anarchial activity of the cerebral centres.

§ 1. PSYCHICAL DISORDERS DUE TO INHIBITION OR DIMINUTION OF MENTAL ACTIVITY

A. NARCOLEPSY.—Pathological sleep or narcolepsy is clearly the most striking and the purest

manifestation of inhibition of cerebral activity. Plunged into deep slumber, the patient loses all contact with the outside world, all voluntary control (*vie de relation*) is lost, and it is only the functions of the vital organs (*vie végétative*) that remain intact.

Everyone has heard accounts of these "trances," and cases are recorded from time to time in the newspapers; but before the war they had become of extreme rarity. In the army, narcolepsy is not very rare. It usually commences suddenly, either during or after a bombardment. The patient falls asleep in a trench or shelter; after a varying interval, attempts are made to arouse him, all without success. From this time the clinical picture is established and does not become modified in any way.

When the limbs are raised they fall back limply or are sometimes slightly hypertonic; respiration is quiet and normal; the rate of the heart is usually slightly slower than normal but the beat is vigorous. The facial expression reflects no psychical activity and resembles that of a tired sleeper. However, on closer examination one is struck by a quivering of the eyelids and a rapid and fine tremor of the orbicularis palpebrarum. If attempts are made to separate the lids, difficulty is experienced owing to contracture of the orbicularis. The eyeballs are usually rolled upwards and immobile. Touching the bulbar conjunctiva calls forth no reflex movement; the same is the case with all cutaneous stimulation, the most severe stimulus not awakening the patient. From time to time he makes movements of deglutition or grinds the teeth. Urine and fæces are sometimes retained, or there may be diarrhœa and incontinence. Sent back, usually with the diagnosis of *coma due to shock,* the patient remains in this condition for several days; then one morning, without any apparent cause, he suddenly wakes. He looks around him in astonishment and declares

that he remembers nothing that has passed since he was under fire.

It is clear that the diagnosis of this so-called hysterical narcolepsy is extremely simple; it ought never to be mistaken for coma due to true concussion, on account of the complete absence of any alteration in the tendon and cutaneous reflexes and the integrity of the circulatory and respiratory functions. The patient always recovers more or less rapidly, according to whether it is left to take its natural course or whether energetic psychotherapy is employed.

Lastly, we must mention that at a Neurological Centre near the front we have only observed one isolated case in eighteen months. Thus medical suggestion and erroneous diagnosis seem to be at work in the fixation of the disorder.

B. The Confusional States.—Just as narcolepsy is characterized by profound sleep, so the confusional conditions are distinguished by a very profound retardation of the psychical functions—torpor, cerebral inactivity, sluggishness of ideation. It is, in reality, a sleep less profound and limited to the functions of the intellect. The confusional states show great variation in their clinical expression; there are all intermediate gradations between confusion with stupor, amounting almost to complete sleep, and simple dulness of the intellectual faculties.

1. **Mental Confusion with Stupor.**—As a result of bombardment and due either to shock or simple emotion, a soldier, who seemed in no way predisposed to such an affection, presents the following clinical picture. Plunged into torpor, with an expressionless face and haggard eyes, he is unable to answer any question. None of the everyday external stimuli produce any reaction. Like the idols of the psalm, "Eyes have they, but they see not; they have ears, but they hear not," and

they seem complete strangers to the world about them. They are easily led and on the battle-field are not influenced by the whistling of bullets nor the bursting of a shell.

The circulatory and respiratory systems are not deranged, and examination reveals no sign of a lesion of the nervous system.

This condition lasts a variable length of time; the return to consciousness is always gradual, the patient passing through the phases of obtusion and torpor, the characters of which we shall study.

2. **Simple Mental Confusion.**—This is essentially characterized by an extreme slowness of ideation, a complete disorientation in time and space, a reduction of the perceptions and a numbing of the emotional faculties—in a word, the slowing-down of all the psychical functions.

Further, memory is affected, and there is always amnesia of a special type, in which exact and precise facts and extravagant forgetfulness are intermingled. This amnesia usually relates to the events which occurred from the moment of the shock and emotion. It is thus essentially an anterograde amnesia or an amnesia of fixation.

As in the preceding form, there is a progressive return to full consciousness; and when the re-establishment of the normal psychical life has occurred, the patient has forgotten the events of the confusional phase.

3. **Confusion and Obtusion.**—Care must be taken to distinguish these one from another; the latter is characterized by disorientation in time and space, the former chiefly by a slowness of ideation and amnesia. In spite of this difference the two conditions may be closely compared, as has been done by most psychiatrists (Régis in particular) on account of their common pathogenesis and mental foundation, dominated as it is by *asthenia*.

Thus, in conditions of obtusion and torpor, the disorientation is not absolute; but the perceptions and apperceptions are retarded and thrown into confusion. The patient appears melancholy and depressed, shuns society, makes no effort to realize what is taking place around him; his slow and laborious replies give the impression of a great effort.

In all these types of confusion, attention and memory are affected; but in certain cases the confusional syndrome is hardly apparent, either as regards the disturbance of attention or of memory. Two special types of mental confusion, in which aprosexia and amnesia respectively are the predominant features, have been described.

(*a*) *Aprosexic Form.*—This form has recently been described by Chavigny and is characterized by the loss of voluntary fixation of attention. The patient is bright and active, the reverse of the condition in true mental confusion. At the mercy of all the various external stimuli, he resembles the infant, who instinctively turns his eyes to anything that shines or makes a noise. As in the infant, it appears as though the sensations are not transformed into perceptions, that they remain isolated and apart from his personality. It should be noticed that in spite of this instability—resembling "that of certain cage-birds which incessantly turn their heads towards noises or movements round about them"—the power of imitation remains unaffected.

This condition persists for several days, after which the patient gradually regains his psychical activity, but he always remains amnesic as regards the events that have occurred since the initial accident (emotion or concussion).

This aprosexia may, as in other forms of mental confusion, be complicated by psycho-neuropathic disorders: mutism, deafness, or convulsive attacks.

(*b; Amnesic Form.*—It is a well-known fact that

amnesia is the predominating feature in certain toxic or infective forms of mental confusion, and it forms the most obvious manifestation of Korsakoff's syndrome. The war has shown that the amnesic form of mental confusion is by no means peculiar to toxic and infective conditions; it has, indeed, proved that in many cases amnesia may be almost the only symptom of confusion due to concussion or emotion; Régis, Chavigny, G. Dumas and the present authors have recorded many very striking examples of this condition. The amnesia seen in the mental confusion of the soldier presents some very interesting peculiarities.

It is sometimes an incomplete amnesia, or more correctly a dysmnesia, a mingling and jumbling of recollection with regard to all that has taken place since the emotion or shock which has caused it; it is "not absolute night but a rather dim twilight." More frequently the amnesia is absolute, a gap or break in the series of recollections. In distinction to the amnesia of toxic or infective confusion, this loss of memory relates not exclusively to the period following the initial physical or psychical trauma, but extends to a period, often quite prolonged, anterior to the accident. It even deletes from the mind of the patient his name, parentage, age and calling. All the previous events of his life may be effaced from memory and cannot be brought back to him. And just as it can be said that the amnesia of toxic confusion is *anterograde* (amnesia of fixation), the amnesia of confusional conditions due to shock or emotion is *antero-retrograde*. We do not apologize for laying such stress on this point, as it is a special character of amnesia due to emotion or concussion. Sometimes from this absolute darkness emerges a solitary recollection—that of the shock; he knows not his name or his age, but remembers having been present at some terrible scene of slaughter. As G. Dumas has pointed out so fittingly, though his memory is in abeyance, he preserves his motor

functions and practical knowledge, which have long been automatic. He is able to dress himself, read, and write.

This antero-retrograde amnesia may be less complete and behave in an extremely curious and contradictory manner. It may affect certain branches of conscious knowledge and spare others.

In some cases it is the visual images which are chiefly affected, the auditory images remaining intact; the patient is unable to picture the most familiar objects, such as a horse or a bird, and he even loses his mental picture of individuals.

These *selective amnesias* may also affect written language and produce the phenomena of aphasia, or, more correctly, of pseudo-aphasia (word blindness, letter blindness). Certain patients preserve the auditory word-image, but have lost the power of graphic representation; thus even the most educated individuals write with the most extraordinary orthography.

§ 2. PSYCHICAL DISORDERS DUE TO INORDINATE EXCITATION OF THE MENTAL ACTIVITY

Hallucinatory Mental Confusion. Oneiric Delirium.—In these cases the etiology is the same as before: emotion and concussion. Mental asthenia, confusion, and disorientation are here not the only symptoms. These conditions are also characterized by *delirium,* usually an active delirium resembling the toxic variety, described by Lasègue as *visionary* delirium, by Régis as *oneiric* delirium. Occurring in soldiers a prey to the daily emotions of trench warfare, it is easy to explain the predominance of some "motif" to do with the war as the usual theme of these manifestations. The confusional delirium, as always, is not confined to the imagination, but is accompanied by gestures; it is not only a dream, but a dream lived through. A prey to oneiric excite-

ment, the patient loses all sense of reality, shows complete contempt of danger, running along the parapet and offering his body as a target, although nothing in his behaviour evinces any emotional reaction. Sometimes, straying at random, he will wander for hours no matter where, then suddenly come to himself, having completely forgotten the initial emotion and shock and all that he has been doing. It will be seen that these confusional escapades are of great interest both from a clinical and a medico-legal point of view. Misconstrued, they may bring upon the patient the most disastrous and undeserved punishment. In the neurological centres near the front-line numerous examples may be found. Such cases may be under trial by court-martial and a mental examination by a specialist ordered.

The hystero-emotional psychoses (H. Claude, Dide, and Lejonne) belong to this group of cases. Here, again, emotion is the basis of these mental disorders, an intense emotion, sometimes accompanied by a feeling of imminent death and maintained by the tragic and terrible scenes of the battle.

The special feature of the psychical condition of patients suffering from these psychopathic disorders is that between the bouts of excitement, no real mental confusion is detected. The patient can speak and reply to questions with great ease. But if one reminds him of some episode in the fight or brings back some recollection or idea associated with it, he is at once overcome by emotion ; he again pictures the scene he has lived through, sees "Boches," throws himself under the bed to shelter from the shells, or runs ahead towards imaginary trenches. The thud of a distant cannon or some metallic noise will often be sufficient to provoke an emotional attack with all the phenomena of oneiric delirium. In such cases the psychological state is characterized by the suddenness of the emotional reactions and their short duration, the absence of systematization

of the emotion and of any dominant delusion and lastly, by the complete integrity of the intellectual processes in their strict sense.

They are purely psycho-neuropathic disorders, analogous to paralysis, contracture and mutism, and as such they recover rapidly, never leaving any sequelæ whatever. There remains only the constitutional condition of emotivity, owing to which the hypnosis of battle (Milian) and the hystero-emotional psychoses can be produced.

It may be a matter of surprise that we have classed in the same group the oneiric delirium of mental confusion and the above conditions. But without entering upon a controversial discussion, we will only mention that Régis long ago pointed out that in reality the oneiric delirium of toxic or infective origin is itself a true somnambulistic condition; that both toxic oneirism and hysterical somnambulism are followed by a retro-anterograde amnesia, clinically identical in the two cases; lastly, that both in toxic delirium and in hysterical somnambulism it is possible by suggestive hypnosis to bring back the lost recollection of the attack.

Thus we consider that the post-emotional or post-concussion form of mental confusion should be ranged in the same class of psychoneuroses as the motor disorders and the affections of the senses and sensation, since they both have the same etiology, the same psycho-pathological basis, the same prognosis, and demand the same methods of treatment.

Diagnosis of the Psychical Disorders.—The identification of the various psychopathic affections we have described is not a matter of much difficulty. The distinction between the various comas and narcolepsy, between true psychoses and somnambulistic delirium, is not very hard to draw.

The differential diagnosis of confusion or oneirism of toxic or infective origin and so-called hysterical confusion or oneirism is not such an easy matter.

As we have shown, Régis has pointed out the clinical analogy.

In actual practice the purely psycho-neuropathic manifestations are most likely to be confused with alcoholic delirium or the organic stupor of those suffering from profound cerebral disturbance.

Since from the purely psychical point of view these conditions are very similar, it is evident that the distinction must be based on the associated phenomena rather than on the mental symptoms. In the case of a toxic or infective delirium, the motif of the delirium itself is less coherent, less systematized (Ballet), and the confusion occurs in the periods intervening between the oneiric paroxysms; it is seldom that some sign of intoxication is not found as the causal factor, generally alcoholism (tremor of the hands and tongue, inequality of the pupils, abnormality of the reflexes and sensation, etc.).

In the case of a patient suffering from cerebral concussion in whom the condition of torpor and stupefaction is due to actual changes in the brain, one will find definite organic symptoms due to disturbance of the central nervous system by the trauma (Babinski's sign, increase or decrease of tendon reflexes, increase of albumin in the cerebro-spinal fluid, with or without leucocytosis). All these have been fully described by Guillain, Oberthür, and du Roselle.

The very course of these manifestations gives a clue to their nature. While toxic symptoms or those due to true cerebral disturbance are slow to disappear, the purely psycho-neuropathic manifestations, so singular in their behaviour, yield with ease to judicious treatment.

We will only mention in passing a mental condition described by Schaïkievitch after the Manchurian Campaign to which he gave the name *amentia depressido-stuporosa.* This is really only a typical mental confusion in which ideas of auto-accusation and persecution are mixed and which, like the confusion

we have just studied, takes origin in an emotion or concussion.

Can we proceed further with the diagnosis and, after clinical examination, define the exact origin of the confusional condition ? G. Ballet and Roques de Fursac maintain that in the "so-called commotional psychoses" the part played by the physical shock is negligible, that by the emotion essential. The principal point on which these authors base their statement is that pure emotion without any physical element can accurately reproduce in form, intensity and course the psychopathic disorders attributed to concussion. G. Dumas does not agree with this view. He points out that cases due to emotion do not lose consciousness and do not exhibit mental confusion until several hours after the incident, while those due to concussion lose consciousness in 95 per cent. of cases.

This statement is correct enough, but, as he himself has observed, though the loss of consciousness may be attributed to the physical shock, this does not exclude the possibility of an intense emotion interposed between the moment of the explosion and the loss of consciousness, an emotion which has unloosed the confusional state.

At any rate we can state that, in the immense majority of cases, confusion due to concussion *plus* emotion (*commotion—emotion*) is not accompanied by any apparent wound and does not present any symptom of a lesion of the brain or cord ; this distinguishes it from the cases of confusion due to concussion (*commotionnés*), which, as we have said, behave quite differently.

It would be going too far to deny that the shock of concussion may slightly affect the brain and thus somewhat reinforce the predominant effects of the emotion.

§ 3. PSYCHICAL DISORDERS IN CASES OF CEREBRAL CONCUSSION

(*Commotionnés cérébraux*)

The purely mental disorders included in the psychoneuroses of war have an almost exclusive origin in concussion and the emotion which it produces. Most physicians who have studied this question have asked themselves which one of these two factors is the real basis of the complaint.

The question is important; for if stress is laid on *concussion* as the pathogenic basis, the necessary conclusion drawn is that there are more or less important changes in the substance of the brain, changes of a unique nature and well known in present-day experimental pathology. The position is quite different if we ascribe the pathogenesis and etiology of these disorders to *emotion.* We are, of course, ignorant of the nature of the changes in the mental elements produced by emotion; but we know that they often disappear very quickly under purely moral and psychical influence, and that if there is any change, it is certainly very different from that produced on the brain by "traumatic" shock.

In point of fact *there are two forms of psychical disturbance following concussion,* one dependent on actual changes in the brain, the other, which may be described as functional and which belongs to the group of psycho-neuropathic disorders.

We by no means infer that the clinical diagnosis of these two groups is always easy; however, in most cases it is possible to draw the distinction.

As we have seen, the immediate mental disorders following concussion are revealed in the various forms of mental confusion, with or without delirium.

These confusional conditions, which often present a clinical aspect betraying their emotional origin and disclosing their psycho-neuropathic nature, may completely resemble toxic confusion, except

that no visceral or nervous symptom can be found to indicate an intoxication. As we have already stated, the differential diagnosis of emotional confusion and that of toxic or infective origin is based more on the associated symptoms than on the character of the mental phenomena. As regards confusion of traumatic origin due to actual lesions, the differentiation of this from analogous phenomena of a neuropathic origin is a still more delicate matter. Moreover, it is quite rare for traumatic lesions of the brain to be followed exclusively by disorders of a purely psychical nature.

Careful examination will reveal changes in the tendon and cutaneous reflexes and in the respiratory and circulatory systems, which are never found in true neuropathic conditions. In addition, if one takes into account the existence of changes in the cerebro-spinal fluid (increase of albumin, leucocytosis, xanthochromia), it is seen that there are a number of signs which will at any rate assist diagnosis. Failing all other methods, the course of the affection will clear up any doubt. Rapid and favourable in psycho-neuropathic confusion, it is, on the contrary, prolonged in toxic and traumatic confusion, leaving *sequelæ*, the origin of which must be carefully considered in view of deciding on the military status of the patient.

These sequelæ are both psychical and physical and are found, with slight variations, in severe cases of concussion. The chief are : persistent headache, especially during the day ; photophobia, labyrinthine and cochlear hyperæsthesia causing vertigo and painful reverberation of sounds, localized or generalized hyperæsthesia, insomnia, bad dreams or nightmares, and lastly, in the psychical sphere, dysmnesia, slowness of ideation, a tendency to depression and melancholia and a highly-emotional condition. The last few phenomena are part of the syndrome described under the name of traumatic neuroses, which we must briefly discuss.

Under the title of *Traumatic Neuroses*, Oppenheim has described a symptomatic picture made up of various elements chiefly of a hysterical and neurasthenic nature, and which he ascribes to slight changes in the elements of the central nervous system, which at present cannot be defined. The traumatic neuroses, therefore, do not constitute a definite pathological group, but rather a complex collection of symptoms, all the details of which we have studied in the foregoing chapters.

But in addition to these symptoms, Oppenheim has shown—and this is the only point of present interest—that very often patients, suffering from what we call concussion, exhibit special psychical disorders of a depressant nature which may reach actual melancholia.

Such neurasthenia-like symptoms are by no means exceptional, and we have often observed them as sequelæ of old-standing cases of cerebral concussion.

The leading features from a psychical point of view are the state of torpor of the psychic activity and the disordered condition of the emotional faculties. Incapable of concentrating on any form of regular work, these patients are ever haunted by the idea of their ailment and the symptoms it has produced.

This constant state of preoccupation leaves its mark on the facial expression, which is melancholy and immobile, always reflecting the constant obsession which hardly ever leaves the mind. They shun society and seem to have lost the power of laughing.

It must be added that most of these patients are tormented by intractable insomnia, or their sleep is always troubled and distorted by dreams of war. These bad dreams and nightmares are often maintained by subjective noises in the ears and by sensory hyper-excitability.

If an attempt is made to stimulate their self-respect, they burst into tears, declaring that they are incapable of undertaking any work and that they

are hopeless invalids. Many suffer from headache, vertigo, dizziness, acoustic and visual hyperæsthesia. Forward flexion of the trunk produces abnormal congestion of the face, vertigo, and a marked slowing of the pulse-rate (Erben's sign). The cutaneous reflexes are normal, but the tendon reflexes are usually brisk.

Changes in the circulatory system are also not of uncommon occurrence: tachycardia when in the upright position (ortho-tachycardia), arrhythmia, instability of the arterial pressure or inequality of the vascular tension in homologous limbs, and dermographia.

The persistence and intractability of the subjective symptoms makes life so difficult to bear that the condition of neurasthenia gets worse and renders the patient a true hypochondriac or melancholic.

The course of this form of traumatic neurasthenia is always protracted and the condition only improves after a lapse of several months.

In spite of the absence of gross objective phenomena, patients suffering from this condition cannot be sent back to the lines; any who, against medical advice, try to return to the front are soon compelled against their will to acknowledge their powerlessness and have to be returned to the base, for a time at any rate.

§ 4. THE MENTAL CONDITION OF PATIENTS WITH CRANIAL WOUNDS AND AFTER TREPHINING

Although a study of the psychical phenomena displayed by patients with wounds of the head hardly comes within the general scope of this book, we think it advisable to mention very briefly their principal characters, which will be seen to resemble the psychical sequelæ of concussion.

The residual psychical syndrome found in patients with cerebral trauma presents, by common consent,

great uniformity. Its constituent factors—especially obvious—are a feebleness of will-power and voluntary attention, slowness of ideation and rapid weakening of the imagination (Villaret and Mignard). Just as in the concussion syndrome we have described above, this torpor of the higher faculties may be accompanied by an oneiric element and by dysmnesia or amnesia, or by the sensory and sensorial hyperæsthesia which is so characteristic of organic conditions following concussion.

Both in cases that have previously been trephined and in cases of severe concussion, it is common to find the signs of *œdema of the optic disc* (Cantonnet), a result of the increased tension of the cerebro-spinal fluid, which we have discovered in a large number of cases and measured by Henri Claude's manometer.

This increased pressure seems to play a part in the production of the purely psychical phenomena observed in cases of severe concussion and of local trauma; it is generally absent in patients suffering from psycho-neuropathic disorders due to concussion. It is sometimes accompanied by increase of albumin and exceptionally by an increase of cells of the cerebro-spinal fluid.

Lastly, the residual nervous disorders in cases of trephining are not only out of all proportion to the wound, but often vary inversely in intensity with the extent of the osseous lesion.

This necessarily brief summary will show clearly the points of resemblance between the concussion syndrome and that observed in cases which have been trephined, so that we are justified in uniting these two conditions from the point of view of pathogenesis. In either case, in addition to the psycho-neuropathic phenomena, symptoms may be found, not in harmony with circumscribed lesions, but rather with diffuse cortical changes and, to some degree, with the increased cerebro-spinal pressure.

§ 5. THE "SINISTROSES" OF WAR

Under the name *sinistrose*, Brissaud has described a psychopathic condition peculiar to workmen's accidents, characterized by a pathological state of restlessness resembling a systematized delirium, based on an idea of unjust treatment.

In some ways the mental condition of the wounded very much resembles that of an artisan injured at his daily work—uncertainty as to his military status, anxiety as to the sick-pay he will receive, etc. Thus we think that Brissaud's expression may be extended to include a psycho-neuropathic disorder quite common amongst our soldiers under the name of the "sinistroses of war."

It commences at a time when the wound is almost healed. The patient is much better and he realizes the fact; but not finding himself as well as he was before being wounded, he fears the return to the front because he will not be so efficient as he used to be. He is generally troubled by pains which do not suggest any lesion of the nerves, but are rather true psychalgia, further increased by concentration of thoughts on the condition of his injured limb. Depressed, suffering, and sleeping badly, he begins to ask himself if he will ever be well again, and if even in the distant future he will be capable of returning to his occupation or to any work whatever.

In short, it is a condition of hypochondriasis, exaggerated and modified by the predominant preoccupation of anxiety as to the outcome of the situation caused by the trauma.

It might be thought that if discharge from the army with a pension be obtained all the symptoms of restlessness would vanish *ipso facto*; as a matter of fact, although complete recovery often follows discharge, this is not always the case. But in these instances the symptoms of the "sinistrose" undergo a change and the hypochondriacal ideas of the first phase are replaced by ideas of fresh claims

and demands. The pure "sinistrose" is followed by a psychosis of demand.

The last condition is too well known to need discussion. We need only note that its development necessitates a soil specially prepared by previous deterioration, and that patients who exhibit such an affection have, since infancy, shown symptoms of mental instability and obvious physical and intellectual defects.

They are egoistical and proud. They soon express ideas of persecution and claim honour and greater recognition. Never satisfied with medical examinations and the decisions of medical boards, they continually protest against the rate of pension which has been granted them, and they use every means to obtain the opinion of nervous specialists, who they say and know will recognize the true nature of their infirmity and secure the pension they deserve.

This psychosis of "discharged claimants" is really only the expression of a more or less latent and constitutional mental condition; it is therefore practically incurable, but the grosser phenomena it displays may be mitigated to a certain extent.

Chapter X

NERVOUS DISORDERS INDUCED BY THE EXPLOSION OF A PROJECTILE AT A SHORT DISTANCE

The Symptoms of Concussion
(*Accidents commotionnels*)

The explosion of a projectile close at hand plays, as we have seen, an important rôle in the etiology of all the neuropathic disorders we have reviewed. In the vast majority of cases it is the origin of the disorder and is quite the most important etiological and pathogenic factor. Therefore, although they differ widely in their mode of expression—possibly also in their nature—the most diverse nervous or psychical manifestations are linked together by a common bond and can be classed together in a common group under the name of concussion disorders or the "concussion syndrome." [1]

[1] Under the name of "syndrome commotionnel" of the traumata of war, Mairet and Piéron describe a syndrome which includes:

(1) Sensory disorders, chiefly hypæsthesia or anæsthesia affecting all forms of sensation—amblyopia, deafness, apraxic amnesia, cutaneous and deep anæsthesia—and special forms of hyperæsthesia (neuropathic *points douloureux*, vibratory hyperæsthesia, osseous hyperæsthesia, etc.).

(2) Motor affections, such as increased reflexes and convulsive fits or functional paralysis.

(3) Vaso-motor and splanchnic affections (especially headache and vertigo).

(4) Emotional phenomena (predominance of sentiments based on the emotions, such as fear or anger, and dissemination of altruistic sentiments).

(5) Associated disorders such as amnesia and apraxia (of which mutism is the commonest type), intellectual inertia, auto natic

Thus the cerebral or medullary concussion of classical description, until now rarely diagnosed and still of disputed pathogenesis, again comes into its own. Its scope is extended to include cases of the most diverse nature, having only the conditions under which they occur as a common bond. The word concussed (*commotionné*) is to-day so commonly used that it has passed into the vocabulary of the lay public, and is even an everyday term amongst the soldiers.

What do we really understand by "concussion symptoms," "commotio cerebralis," "nervous shock"? What is the nature of these disturbances? Are they symptoms of a physical or organic type, caused by the shock or by the atmospheric vibrations of the explosion? Or are they disorders solely of a dynamic or functional nature? In either case, can we discover the "why and wherefore"? Such are

hyper-excitability of the imagination (nightmare, hallucinations, hallucinatory delirium).

(6) Lastly, disorders of the function of apprehension (amnesia of fixation, incapability of fixing the attention).

We do not adopt Mairet and Piéron's description, which seems altogether too cumbersome and inexact. One or more of the phenomena described may, of course, be observed in shock, but the whole series is never present in one case. Thus, it hardly represents a clinical syndrome in the strict sense of the word, but rather an enumeration of the series of psychoneurotic disorders that these patients may display.

Clovis Vincent distinguishes three categories:

(*a*) Emotional symptoms.
(*b*) Symptoms of concussion.
(*c*) Mental symptoms.

He regards these as three types which each confer special and distinct characteristics on the patient.

The man who is suffering from emotional symptoms is one who has not lost consciousness; he is able to flee from the cause of his fear.

The man under the influence of concussion-phenomena becomes completely unconscious at once, and remains so for a fairly long period. He remains near the spot where the explosion has occurred and has to be carried away.

The man exhibiting mental disorders is like neither of these. He

the questions which we are going to try to answer in this chapter, given over to a summary, having before us the large number of facts which neuro-psychiatric observation has supplied during the present campaign. We are here in touch with a most interesting problem, both on account of the theoretical questions which it raises and the medico-military importance it presents. This interest is shown by the many discussions which have centred round it; one by one, partisans of both the organic and inorganic theories have been compelled to recognize a single, unique cause as the basis of these "commotional" disorders.

As a matter of fact, if the points at issue are catalogued and every preconceived idea put aside, on final analysis it is found that the differences of opinion are, perhaps, more apparent than real. It

is not unconscious, as he stands up, walks about, sometimes shouts and struggles. He is not a prey to emotion, for he is often mentally inert and affected by complete mental confusion.

This seems too rigid a classification, and does not accord with the observations which Boisseau and one of the present authors have made in a very large series of cases in a Field Neurological Centre during offensive operations.

We found that the emotional, concussion and even the mental types were generally not sufficiently distinct enough to allow of their differentiation.

Lastly, Georges Guillain has described a syndrome following the explosion of a large projectile without causing an external injury, which he describes as follows:

The patient suffers from profound asthenia, which necessitates rest in bed for the first few days; walking is impossible, but later can be performed, though with ataxia, even of the upper limbs. The physical asthenia is accompanied by a psychical asthenia: all the psychic functions are slow (memory, association of ideas, etc.). There is often tremor of the fingers, lips, etc., and dizziness with phenomena of voltaic vertigo. Changes in the tendon reflexes and bradycardia or tachycardia may be found in the early stages. Lumbar puncture shows that there is no alteration in the cerebro-spinal fluid.

The elements of this syndrome may or may not be associated with direct lesions (auricular disorders, deafness, etc.).

These nervous affections tend to progressive recovery.

is owing to a misunderstanding of the exact meaning of the terms *concussion, organic lesion,* and *functional* disorder, and also according to where the cases are observed (front line, base, etc.), that these differences of opinion have arisen.

§ 1. Clinical Description of Nervous Concussion, Commotio Cerebralis. Nervous Shock

These make their appearance in soldiers of every age, in the younger classes just as in the home-service men of riper years, in men with previous constitutional neuropathic traits or with such tendencies acquired during the campaign, and in which hyperemotivity plays a prominent part; lastly and more rarely, in men without any predisposition. Sometimes the shock is observed as a result of the first bombardment in a man who has only recently taken his place in the trenches; sometimes, on the contrary, in men hardened to shell-fire by many months of fighting. We shall repeat here all we have said with regard to the origin of nearly all the other psychoneuroses of war.

The predominant factor, however, in the causation of nervous concussion is the determining rôle of the various violent explosives—subterranean mines, aerial torpedoes and mine-throwers, large projectiles which, with a tremendous din, cause a violent displacement of air and of soil. Shock is also observed, but much less commonly, following the whistle of shot alone or the rattle of the 77's.

From a clinical point of view, the symptoms of concussion (commotional symptoms) may be arranged in three chronological groups, according to the time and place where they are observed:

(1) *Immediate Symptoms,* of the battle-field and front-line trenches.

(2) *Early Symptoms,* in the field-ambulance or field-hospital.

(3) *Late Symptoms,* in the general hospitals at the

base and the neurological centres of the interior. The two latter, with slight distinctions, often merge into one another. It is obvious that this classification is quite arbitrary, varying with the military position, which necessarily modifies the system of evacuation. It has the merit, however, of pointing out the variation of the clinical aspect according to the place of observation, a point which has not received sufficient attention. Just as in the surgery of the war, there is a neurology of the front-line and a neurology of the base.

1. *Immediate Symptoms.*—Convulsive attacks or epileptiform fits are seldom witnessed under the fire of the enemy, but when seen, they occur in the depths of a trench or in a sheltered position; they often commence at the onset of a bombardment, while a position is being established.

When there is an advance, the patient runs to the rear-lines and makes straight for a first-aid post or dressing-station.

To quote from du Roselle and Oberthür:—"On a certain day of violent bombardment, several men suffering from 'shock' come rushing pell-mell to the first-aid post; they have no apparent wounds, but are haggard, and stagger as they rush in; not one of them can reply to any question that is put to them, and they recognize nobody. One of us approaches the man who seems in the worst plight and tries without success to bring him to his senses, then leads him by the arm to the first-aid post so as to examine him at leisure. Just as the patient is going to pass in, he notices the recent marks of a shell which has penetrated the wall. He immediately draws away and starts to argue—'No, Major, I don't want to go in there, it is under shell fire now; it wasn't so yesterday'—and he cannot be made to advance a step. Left to himself, he and his unfortunate comrades lie prone in a little blockhouse that has been built to shelter wounded

during the bombardment, pending an opportunity to evacuate them. These men lie there trembling and cowering, not replying to any question and apparently incapable of any voluntary act. There is no need to explain to them when the vehicles arrive; they understand at once; they climb in without help, perfectly naturally. The journey is accomplished without incident."

These symptoms are very different from those which mark direct concussion of the central nervous system, the result of the shaking of the cerebro-spinal axis by the patient being thrown to the ground, against the trench-parapet, or, as frequently happens, by temporary burial.

This direct concussion finds its extreme expression in the sudden death which, as we know, is unfortunately not uncommon.

Grave cerebral disturbance is shown by immediate loss of consciousness and complete coma of the cerebral type, with muscular relaxation and abolition of the cutaneous and tendon reflexes. The limbs are in a hypotonic condition. Babinski's sign may even be obtained, and also Pierre Marie and Foix' contraction sign with an exaggeration of the defensive reflexes.

In less severe cases only a transient loss of consciousness and amnesia are observed; the man is able to get to the first-aid post, held up by the stretcher-bearers.

If examined at once the face shows special characteristics: labial commissures depressed, paresis of the tongue, ptosis with external opthalmoplegia, mydriasis, and inequality of the pupils and nystagmus.

As in the previous form, the muscles of the limbs and the trunk are hypotonic and the patient cannot either stand or sit without assistance. When lying down, he often makes asynergic and clumsy gestures.

In spite of their dramatic appearance, which would justify a very reserved prognosis, these symptoms

of shock die away quite rapidly and do not call for an immediate evacuation to the base (du Roselle and Oberthür).

2. *Early Symptoms.*—These are the symptoms observed some hours or days after the trauma, in the various hospitals at the front, and particularly in the army neurological centres. We shall only briefly recapitulate them, on the basis of the numerous cases that have come under our personal observation, as they have all been studied in detail in the first part of this book.

From a purely clinical standpoint, setting aside for the time being the question of pathogenesis, the cases of shock which arrive at the field-ambulance present three principal types; these, needless to say, are sometimes merged one into another.

A. **Shock of Psychopathic Type.**—This is the most common. The symptoms point to a condition of mental confusion in its slight, moderate, or severe forms: *simple mental confusion,* cerebral dulness, dysmnesia, disorientation in time and space, with constant and often intractable headache and occasionally a rise of temperature; *hallucinatory mental confusion* (oneiric delirium); and *stuporous mental confusion,* with torpor, mutism, and pronounced lethargy and stupefaction. These may be accompanied by psycho-sensory disorders (deafness or deaf-mutism) or by psycho-motor affections (convulsive attacks, tremor, paralysis, etc.); but the dominant feature of the clinical picture is the psychical disorder—*mental confusion*; this it is that determines the treatment.

B. **Shock of Neuropathic Type.**—In this group we include the patients who exhibit predominant symptoms of a purely neuropathic type, without any trace of a confusional state at the time of observation. The latter may, of course, have existed

some hours previously in the form of hebetude or incomplete obtusion which has disappeared, leaving nothing more than slight amnesia or lacunar dysmnesia (failure to bring to mind the cause of the trauma). In other cases there is no appreciable external disorder, the patient has not lost consciousness and he can give a fluent and detailed account of the accident which brought him to the field-ambulance ; if he cannot speak, he writes an account of it. This is why we find it difficult to agree with Dumas that a confusional condition is necessarily the basis of all neuropathic disorders following concussion. In short, we have here examples of the classical type of *traumatic psychoneurosis* or *traumatic hysteria.*

Any one of the long series of symptoms described in previous chapters may thus be encountered ; we will only enumerate them here in the order of frequency with which we have observed them : —affections of hearing and speech (deaf-mutism, deafness, mutism, aphonia, stuttering), tremors, choreiform movements, convulsive attacks, paralytic motor disorders (monoplegia, paraplegia, hemiplegia), contractures (curvature, acro-contractures), disorders of the gait (astasia-abasia, staso-basophobia), diffuse or localized pain, affections of the sphincters, and lastly, neurasthenic or psychasthenic conditions.

Abnormalities may or may not be found when the reflexes are tested or the cerebro-spinal fluid examined. The tendon and bone reflexes may be normal, feeble, or more often rather brisk but not really exaggerated ; the cutaneous reflexes show the same characters. As regards the cerebro-spinal fluid, increase of albumin may be present (Ravaut, Guillain, Leriche), but is rare (Roussy and Boisseau, Claude), and lymphocytosis is quite exceptional ; on the other hand, increased pressure is frequently observed, tallying with the headache, when lumbar-puncture is a valuable method of treatment.

These observations with regard to the cerebro-spinal fluid apply to patients of both groups (*A* and *B*). In none of these cases, apart from the increased pressure and slight increase of albumin in the cerebro-spinal fluid (which is not constant and is of very debatable semiological value), is any single feature discovered which might point to an organic change in the nerve centres.

Clinically, on the other hand, they all fall into the group of psychoneuroses which we have defined at the commencement of this volume.

C. **Shock of Organic Type.**—This third group includes the cases in which the concussion is followed by signs of lesions of the brain, cord, or meninges. These are clear, indisputable, and more or less permanent signs, shown by disorders of motor, sensorial, sphincteric, or reflex nature, and by changes in the cerebro-spinal fluid—changes in the cells or proteins and xanthochromia—due to meningeal or cerebral hæmorrhage, hæmatomyelia, encephalomalacia or myelomalacia, etc. Whether these disorders be slight or severe, they persist either permanently or, at any rate, for a long period, and thus stand out in contra-distinction to the two preceding groups, which are always transient and quickly cured. .

3. *Delayed or Chronic Symptoms.*—These are the psycho-neurotic sequelæ, which concussion symptoms leave behind them. Besides all the nervous effects described in the preceding paragraphs—symptoms which are seen as often at the base as at the front—these are some which by their chronicity little by little change their clinical type. We refer to the *nervous sequelæ following concussion* and the *sinistroses of war.*

The former are common, and the long duration of the war has brought them into prominence. They consist of headache which is often intractable

and unrelieved by analgesic drugs, and particularly of disorders of a psychopathic nature: such as change of character, persistent dysmnesia, the psychasthenic state, excess of emotion, and various forms of terror and fear, which cause men previously brave and courageous to lose all self-control and make them unable to take their place in the trenches, which until then, and during all the campaign, they had occupied without flinching. If they are sent back to the firing-line, the slightest cause produces a relapse. They are true "psychical invalids." These facts are valuable from the point of view of deciding upon the right course to pursue.

Of still greater importance is the "fixation" of neuropathic symptoms. The patient acquires a peculiar mental state which, by analogy with that described by Brissaud in workmen's accidents, has been described under the name of "Sinistrose of War." We have already discussed this condition (p. 849).

§ 2. Nature of the Nervous Concussion (*Commotion*)

We have already observed that the pathogenesis and nature of the symptoms due to concussion have been much discussed during the last two years. A rapid review of the publications that have appeared will show us the different phases through which this subject has passed.

Ravaut, Heitz, Guillain, Sollier, Chartier, and others have attempted to ascribe most of these symptoms to an organic lesion of the central nervous system, a lesion induced by atmospheric decompression, the result of an explosion.

Then there came a reaction to this view, when it was shown (Roussy and Boisseau, Claude and Lhermitte) that these central organic lesions, far from being frequent, are of exceptional occurrence, and that in most cases the symptoms of shock are

due to an inorganic, functional, and transient disturbance which can rapidly be cured by psychotherapy.

Lastly, at the debate which took place recently at the Neurological Society of Paris (April 1916), it was decided that a division must be made and that two varieties of concussion symptoms exist, which differ as much in their cause as in their nature. There are in reality cases where at the moment of the explosion, soldiers are contused by the giving way of the ground and the throwing up of earth —*direct concussion*, and other cases in which nervous symptoms appear which cannot be traced to external trauma—*indirect concussion* (Claude, Roussy).

A. Direct Concussion—Evidence of Contusion. —It often happens that at the time of explosion of an aerial torpedo, a shell, or especially a mine, men are buried by the giving way of a trench-parapet, a blockhouse, or a dug-out, or are struck by fragments of earth or stone, or may be thrown violently to the ground. As well as being concussed (loss of consciousness), they are bruised. These facts often remain unobserved, since the patient himself can give no information of the course of events. He honestly believes that it was the atmospheric disturbance of the explosion that caused the shock; or he may have no views on the subject, as he has lost consciousness and suffers from retrograde amnesia as regards the explosion itself. But if careful inquiries are made of the occurrence, if necessary from his comrades who saw what happened, the above causation will prove correct. Now it is under such circumstances as these that the immense majority of cases of shock due to organic lesions occur, arising from areas of softening or hæmorrhage in the brain or cord, or meningeal hæmorrhages.

In short, the displacement of air and escape of gas from the explosive only have a secondary, indirect action on the nerve centres; it is this that causes the fall and burial of the men and accounts

for so much external contusion, especially of the head and spine. It is the indirect shaking of the central nervous system which produces areas of softening or hæmorrhages. Claude and Lhermitte have recently made careful anatomical and clinical observations of these still little-understood cases of indirect ischemic lesions of the central nervous system due to trauma of the neighbouring structures. In any case, such lesions cannot possibly be caused by changes of atmospheric pressure and cannot be identified with those produced by caisson disease.

We must repeat that such lesions are rare. The shock is followed usually at once by grave psychical phenomena—amnesia, severe headache, vertigo, etc. For the rest, there are the classical signs of cerebral concussion, fully known and described before the war.

Lastly, it is possible, but must be extremely rare, that contusion may be produced by the atmospheric disturbance alone, acting as an external shock, particularly in enclosed spaces.

B. Indirect Concussion—Evidence of Disturbance.—In these cases, on the contrary, no external shock is in question, but it is a matter of indirect concussion at a distance.

When the patient is questioned (if he has not lost consciousness), this comes out very clearly. And frequently—as we have ourselves seen and must again insist on—it is only after a lapse of time, after safe arrival at the first-aid post, that the symptoms of shock come to the surface. It is to this category that the psycho-neuropathic disorders belong, the nervous phenomena with which we are specially concerned and which may assume the most diverse symptoms, and numerous forms of which have been described. The rapid recovery under psychotherapy is a clear proof of their neuropathic nature.

The pathogenesis of these disorders is a psychical, not a physical, disturbance; the morbid condition

is thus ultimately ascribable to an emotional shock and the various psycho-pathological factors which we have already discussed.

To sum up, we see that there are two great varieties of nervous shock, differing from one another both from a pathological and anatomical point of view :

1. *Symptoms of contusion* (direct concussion), dependent on a lesion of the central nervous system, which may be circumscribed (rare) or diffuse (common); this is produced by a direct physical jarring upon the osseous walls of the brain and spinal cord (fall, trauma, etc.), and in which the atmospheric disturbance of the explosion only plays an indirect part.

2. *Symptoms of disturbance* (indirect concussion), which depend on a purely psychical pathology (psycho-neuropathic disorders), and are not accompanied by any known signs which could point to an organic disturbance of the central or peripheral nervous system (Roussy and Boisseau).

In the latter group may we not be dealing with minute cellular or fibrillary lesions of the nervous centres ? This is possible but not demonstrable. Viewed in the light of our present knowledge it is quite impossible to say what anatomical or dynamic changes answer to the various neuropathic or psychopathic conditions.

SHELL-SHOCK

As the result of exposure to exploding artillery projectiles, soldiers who are not struck by the bursting shell may nevertheless suffer an acute impairment of their general health, with very serious manifestations, especially soon after the accident. In exceptional cases, death may occur under symptoms of syncope, the body presenting no visible traces of injury. These nervous symptoms in soldiers who have been exposed to shell-fire without suffering demonstrable physical injuries, are popularly and conveniently, but not correctly and satisfactorily summed up under the term, "shell-shock." The condition represents a "war-neurosis," an outbreak of neuro-psychological disease under the stress and strain incidental to modern warfare, notably sustained shell-fire with high explosives. The underlying psychology of the war-neuroses can here be only cursorily reviewed.

In its gravest form shell-shock means death, which sometimes occurs in the absence of any evidence of traumatism, and is possibly due to injury of the central nervous system. A number of the non-fatal cases of shell-shock are characterized by a peculiar neurological symptomatology, suggesting a mechanical source of origin. The symptoms of shell-shock may be those seen in traumatic neuroses. Here the possibility of physical or chemical changes of the central nervous systems cannot be positively excluded. There are also cases, however, in which the contingency of even the smallest damage of the central nervous system through the direct effect of explosives is remote, the patient having shared his surroundings with many others who remain entirely free

from symptoms of any kind. Among seriously wounded soldiers, hysterical shell-shock is very rarely seen.

Shell-shock includes no new or baffling disorders. The condition is characterized by disturbances of sensation and motion. In their recent monograph on shell-shock and its lesions (Manchester and London, 1917), G. Elliot Smith and T. H. Pear point out that a soldier may be struck blind, deaf or dumb by a bursting shell; in rare cases he may exhibit all three disorders simultaneously or even successively. These troubles often vanish after a short space of time as suddenly and dramatically as they appeared. "The muscular system may be affected in an equally striking manner. Contractures often occur in which a man's fist may be immovably clenched for months, or his back may be bent almost at right angles to his lower limbs, there being in neither case any bodily change discoverable by the neurologist which can account for such a condition. These contractures, though curable, often prove very obstinate, and at present their nature remains somewhat of a mystery. Other distressing and persistent disturbances are muscular twitchings, tremors or loss of power in the limbs.

"Not every nerve case, however, presents such striking and objective signs as those which we have just been describing. The subjective disturbances, which are apt to go undiscovered in a cursory examination of the patient, are frequently more severe than the objective, and are experienced by thousands of patients who to the mere casual observer may present no more signs of abnormality than a slight tremor, a stammer, or a depressed or excited expression. These afflictions: loss of memory, insomnia, terrifying dreams, pains, emotional instability, diminution of self-confidence and self-control, attacks of unconsciousness or of changed consciousness sometimes accompanied by convulsive movements resembling those characteristic of epileptic fits, incapacity to understand any but the simplest matter,

obsessive thoughts, usually of the gloomiest and most painful kind, even in some cases hallucinations and incipient delusions—make life for some of their victims a veritable hell. Such patients may have recovered from sensory or motor disturbances and yet may suffer from any or all of these afflictions as a residuum from the original shock-complex."

Four factors have been held responsible for the condition known as shell-shock: (1) Air-pressure, in form of condensation and rarefaction of the air, or rapid alternations of air-pressure; (2) poison gases, with or without simultaneous internal burns; (3) mechanical concussion of the entire body, especially the brain and the sympathetic system; (4) psychic emotion. The important bearing of the psychic attitude on the sequelæ of explosions is illustrated by observations upon sleeping soldiers, in whose close vicinity shells and bombs have been known to explode without producing nervous or psychic symptoms of any kind. Superadded to a congenital nervous or psychopathic constitution are the aggravating factors of loss of strength, lack of sleep, insufficient food, overstrain of soul and body, with great moral responsibilities and a growing horror of the war itself. Psychic resistance is gradually undermined, and the culminating acute terror caused by the bursting shell results in the onset of "shell-shock." The physical manifestations are identical with those of traumatic hysteria and can be explained as due to a disturbed contact between mind and body, at the instant

Electricity in the Treatment of Shell-Shock.—For shell-shock of the neurasthenic type, cerebro-spinal galvanism was found successful according to Captain Wilfred Garton (Brit. Med. Jour., 11, 1916, p. 584); assuming that the condition is due to a paralysis of the nerves regulating nutrition, or a disorder of metab-

olism. Electricity in these cases may be expected to stimulate the affected nerve centres.

"The best form of apparatus is a battery of wet Leclanché cells connected to a switchboard on which are two resistances, each of 1,500 ohms, one in series and the other in parallel with patient and milliamperemeter. The treatment is then commenced with no resistance in parallel and full resistance in series. The parallel resistance is first increased to its full extent and then the series resistance diminished until the required amount of current is obtained. If this apparatus is not at hand, a battery of twelve dry cells with a cell collector and galvanometer, and a resistance of at least 1,500 ohms may be used. No current derived from the mains or any universal apparatus should ever be used for this treatment. A pad composed of about sixteen layers of lint, soaked in a solution of sodium salicylate in distilled water, is applied to the forehead, care being taken that the pad is evenly wetted, and that its centre coincides with the middle line of the forehead. Over the centre of the pad is placed a metal plate, and the whole firmly bandaged to the head. The metal plate is connected to the negative pole. A pad of the same thickness about 10 inches by 6 inches is soaked in tap water, covered with a metal plate, and firmly bandaged to the lumbar region. This is connected to the positive pole. The current is increased very slowly, taking about twenty minutes to attain the maximum of 20 milliamperes, at which point it is allowed to remain for twenty minutes, and then is slowly reduced to zero.

"When increasing the current, the cell collector is advanced before reducing the resistance, and when decreasing the current the resistance is used first."

For a first treatment, Garton gives only 10 milliamperes for ten minutes, and does not pass the full strength of current (20 milliamperes) till the third or

fourth treatment. With nervous patients, labile galvanism may be applied to the spine for two or three days before commencing cerebro-spinal galvanism.

Suggestion—Treatment of Shell-Shock.—Suggestibility is known to be greatly increased in patients partially under the influence of chloroform. Milligan (Brit. Med. Jour., 11, 1916, p. 731) advises treatment by suggestion, conducted in a quiet room apart from other patients. Chloroform is slowly administered and suggestion carried out by the anæsthetist when the patient has reached the required stage, and should be continued until consciousness is fully restored. The after-treatment consists in prolonged rest and change of surroundings.

Isolation of the patients, suggestion and hypnosis, manual work as a therapeutic measure, all have their advocates in the management of nervous and mental disorders developing in connection with "shell-shock."

Adrenalized Serum.—This treatment yielded excellent results, according to Dupont (Archiv. de Méd. et de Pharm. Milit., XLV, No. 5, 1917), in even the gravest cases of traumatic shock, whether or not preceded by profuse hemorrhage, also in wounded soldiers weakened by protracted suppuration.

Mode of Application.—Morning and evening in the first two or three days, an injection is applied of 500 g. of saline solution to which has been added ½ milligram of adrenalin. With this total dose of 1 mg. daily, the small or missing pulse becomes full and strong, and the general condition improves. Operative intervention becomes possible with good prospects of success. Continued administration of adrenalin is not desirable, and after two or three days the treatment should be interrupted and resumed after a variable interval, according to the condition of the pulse. Saline alone, or with the addition of camphorated oil, caffein, or strychnin, even in large doses, is not as active as adren-

alized saline, but all these may be used to supplement the adrenalin when this remains inefficient.

Lecithin was advantageously employed by Lond (British Med. Jour., 11, 1916, p. 168) in the treatment of shell-shock and nervous exhaustion. It is most conveniently administered in capsules containing three grains.

Sequelæ of Shell-Shock.—This experience has been held responsible for defective vision, the ocular symptoms being presumably due to peripheral (choroid or retinal) lesions rather than to concussion of the brain or psychic traumatism. Functional blindness is more common than deafness, loss of smell or taste. Auditory hallucinations and an exaggerated sensibility to sound are characteristic of a number of the cases.

Ocular and aural manifestations only exceptionally appear as isolated symptoms, usually forming part of a more or less extensive symptom complex. In a number of patients there is loss of memory, confusion, and mental depression.

For the treatment of shell-shock and its neurological sequelæ—which is largely one of psycho-analysis and re-education—the establishment of special hospitals has been found necessary by the military authorities, both in France and in England. The Red Cross Military Hospitals, Maghull, Liverpool, organized and superintended by Major R. G. Rows, and other special institutions are specially devoted to the treatment of soldiers suffering from "shock" and other psychoses, where the treatment of nervous and mental disorders in the early stages is very successfully handled. Treatment is conducted upon general lines, rest, feeding, indoor and outdoor recreation, and massage in suitable cases. At the Maghull Military Hospital, a form of psycho-analysis has been used with benefit in selected cases. The results of treatment at Maghull show about 40 per cent of cases returned to light duty. (W. A. Turner, Lancet, 1, 1916, p. 1073.)

Chapter XI

GENERAL ETIOLOGY

In the various chapters dealing with the individual psycho-neuropathic conditions we have already indicated the etiological factors at work. We must now briefly return to these from a more general point of view and discuss the causes which are common to all the psycho-neuropathic disorders.

The chapter on the general outline of the psychoneuroses also contains a résume of the mode of action of the determinant factors, such as we understand them to be.

§ 1. Predisposing Causes

Since predisposition should be considered in discussing the etiology of every disease, this is doubly necessary in the case of the psychoneuroses, where it occupies such an important place.

Although its scope is difficult to define with accuracy, it is quite certain that a *neuropathic constitution*, hereditary or acquired, is a potent factor in the causation of these disorders.

This constitutional tendency does not remain absolutely latent, like that of the psychoses, which suddenly appear without any previous premonitory signs ; but it shows itself, unobtrusively it is true, in certain circumstances of life, which bring into play the emotional reactions of any one of us.

This psycho-neuropathic constitution of which we are speaking seems, if not the most commonplace, at any rate the most frequent. It may reveal itself by two modes of expression : those of an *emotional*

nature, an exaggerated expression of the psychical and physical reactions of the emotions, and those of a *hysterical nature,* the essential characteristic of which is the faculty of *dissociation of the psychical functions* and the *prolonged "fixation" of the disturbance of a function.*

As we have pointed out, careful cross-examination of patients suffering from a psychoneurosis will reveal a latent tendency to these abnormal manifestations of an emotional or hysterical type, which the intense emotions evoked by war have considerably increased and rendered actually pathological.

Thus we believe that of the patients exhibiting psycho-neuropathic affections, the vast majority are predisposed; this predisposition may be hereditary or constitutional, or date from childhood as the result of lack of the necessary education.

It is clear, however, that constitution is not the sole predisposing factor. We must not overlook the causes which make for an enfeeblement of psychological "tone," many of these dependent on the war itself.

The depressant action of *physical or mental fatigue* is undeniably at work; it is a matter of common knowledge that psycho-neuropathic disorders frequently occur after a prolonged bombardment, which has demanded a considerable expenditure of energy.

Further, although it plays a more modest part, we must not omit to mention the *temporary shortness of food,* which often occurs in an isolated body of men to whom supplies cannot be sent and who have to live on their reserve-rations. The factor of *starvation* has always been considered as an important element in the psychoses of war; we do not think that any of the allied troops in the West have ever suffered from starvation, but it is true that certain units have for a short time had to exist without fresh food and drink. This shortage of food may have had its effect in the production of neuropathic and delirious symptoms.

To this shortage of food and drink we must add the various *intoxications*, in which *alcohol* takes first place.

We are inclined to lay too little stress on the results of alcoholism not only amongst troops behind the firing-line, but also amongst the actual combatants. Fatigue and lack of occupation partly account for the " charm of the cup."

Although excess of alcohol but rarely leads to the commoner symptoms of alcoholism, it is a potent factor in the promotion of psycho-neuropathic disorders. As is said of tuberculosis, we may also say that in many cases alcohol " prepares the bed for the neurosis."

We ourselves have observed many cases demonstrating the connection between alcohol and hysteria both at the front and at the base. In many cases one has seen a fit of hysteria major follow a slight excess of alcohol.

We cannot lay too much stress on the danger of abuse of alcohol, not only because of the toxic conditions which are its peculiar property, but also because intoxication, by damaging the central nervous system, makes it so much the more sensitive to the emotional shock of war and causes it to react to an excessive and extravagant degree.

The influence of other toxic conditions—especially excessive smoking—is much more difficult to assess, owing to frequent association with alcohol. We cannot find any real evidence that it increases the toxicity of the latter.

Is there any etiological factor to be found in the *previous occupation*, the *social status*, or the *military position* of the soldier, favouring the psychoneuroses ? Without being able at present to give a very precise answer, the few facts in our possession, apart from our personal observations, allow us to give a few general indications.

Previous occupation and social status in themselves do not militate for or against these complaints.

We have observed neuropathic disorders in men in almost every walk of life.

Nevertheless, the professions which require the exercise of judgment and criticism, and which demand a sustained effort, both physical and mental, not only do not favour the occurrence of neuropathic affections, but clearly militate against them. H. Claude has observed that it is rare to find such disorders amongst skilled officers, especially medical officers.

It is true that in the case of an officer, it is not only his critical faculties and powers of judgment that are at work in holding the emotional reactions in check, but also the knowledge of his responsibility and the good example he must always show. The officer is not a unit which may be lost in a large group, but he has an individuality on which all eyes are focussed, and his own *amour propre* prevents him from " flinching " under any pretext whatever.

This mental steadiness of the officer has a bearing not only on the individual himself, but also acts as a stimulus to the soldiers under his command. Thus, as several officials in the medical services (Dide, Clunet), have pointed out, the infrequency of such affections in the *troupes d'élite* is accounted for by the stimulant influence of the high morale of the commanding officers.

§ 2. Exciting Causes

We have already seen that the four principal determining causes of the psychoneuroses are *concussion, local trauma, emotion,* and *suggestion.*

Shell concussion may be direct or indirect. By *direct concussion* we understand any pathological condition of the central nervous system caused by physical shock affecting the osseous walls that surround the encephalo-medullary axis. Whether the individual has been thrown to the ground, into a trench or against the wall of a dug-out, or whether

a shell, bullet, or any other object has struck the skull or vertebral column, the result is the same : a shaking of the nervous system.

Indirect concussion is quite another matter. If there is any actual disturbance of the cerebro-spinal system, it is produced by atmospheric vibrations which are transmitted to the brain and spinal cord by the movements of the cerebro-spinal fluid, which receives either the effects of the tympanic disturbance or the sudden change of arterial tension.

Direct concussion, though a cause of psycho-neuropathic disorders, often produces organic lesions of the nervous system ; indirect concussion, on the contrary, produces neuropathic symptoms practically exclusively.

What share does *emotion* take in the etiology of psycho-neuropathic disorders ? This is a question which at present cannot be answered with accuracy. A glance at the discussion which took place at the Societies of Neurology and of Psychiatry in 1909 will suffice to show how divergent the views of various authorities really are.

Has the war solved the question by bringing any really demonstrable evidence to bear ? We think not, at any rate up to the present.

We are strongly of opinion, and have already insisted on the point, that the rôle of the emotions in the determination of the psychoneuroses is fundamental and primordial. Much more than shock pure and simple, emotion is itself able to produce a neurosis or to localize and fix on some part of the body the psychological disorder, which is the very basis of the neuropathic phenomenon.

The reply may be made that frequently concussion and emotion are both at work and that it is almost impossible to separate these two factors. We cannot agree with this. Concussion pure and simple, unaccompanied by any emotional element, rarely gives rise to neuropathic symptoms. The latter

may certainly follow concussion; but either the shock has only been relative and not entailed loss of consciousness (and thus may have been accompanied by emotion), or the neuropathic manifestations have not been produced immediately but only after an intercalary phase of retrospective emotion or suggestion.

Local trauma must be considered along with general concussion.

It plays a most important part; it is not only capable in itself of creating a neuropathic manifestation in a predisposed individual, but it also constitutes the agent of fixation or crystallization of a neuropathic condition which, until this time, has been latent and indefinite.

Local trauma acts in various ways in producing marked alterations in the motor and sensory functions of some region of the body. It acts first by creating an *emotional state* which throws its weight and intensity into the circumstances of the combat; secondly, by suggesting to the mind of the individual, under cover of the emotional mental asthenia, the idea of a disorder of some bodily system far greater than any that could be caused by the organic mischief produced by the trauma.

It is known, for example, that shock to a limb caused by a projectile of high velocity may immediately produce a "stupor" of the nerves and a transient, but complete motor and sensory paralysis. In a normal individual this torpor and paralysis disappear rapidly, within the space of a few minutes. It is quite another matter in the neuropathic subject, in whom this impotence becomes fixed and made stable by the action of *auto-suggestion*.

To sum up, we have seen that the two chief causes of a neuropathic disorder or psychoneurosis are first, *emotion* or the revival of an emotion, and secondly, *auto-suggestion*, facilitated by the condition

of mental perturbation originated and created by the emotion.

As regards *exogenous suggestion*, this is frequent, but plays, we think, a more modest rôle than auto-suggestion. *Medical hetero-suggestion* is the most important form. Its influence is most clearly evident in creating disorders of sensation (anæsthesias of various distribution), but it may also be the origin of motor affections. Thus care must be taken never to allow a patient to see or hear a diagnosis of organic paralysis, unless it is absolutely definite and established. The idea of incurability is the more difficult to eradicate from the mind of the patient, the more competent the individual who has made the diagnosis.

Hetero-suggestion from patient to patient is described by all neurologists, ourselves included, as being very rare.

It is possible that hetero-suggestion may play some part by means of the tales and confidences that pass between the patients and which so rapidly spread. We cannot deny the possibility, but its rôle cannot be compared with that either of emotion or of auto-suggestion.

Chapter XII

TREATMENT OF THE PSYCHONEUROSES OF WAR

The treatment of the psychoneuroses of war depends directly on their nature; being disturbances based upon a psychical foundation, they yield to a rational and persuasive psycho-therapy, such as has been recommended and practised for many years by Dejerine, Dubois, Babinski, and others. As Brissaud puts it, "It consists of the employment of means destined to demonstrate to the patient his will-power and to train what is left of it in the best possible manner." This method has beneficially replaced the old methods of hypnotic suggestion which ought definitely to be rejected.

But the war having infinitely multiplied the number of the psychoneuroses in a relatively short space of time and modified their form of appearance, ought also to bring forth a correspondingly energetic and rapid method of treatment. There is the need for coping with the spreading of neuropathic disorders by mental contagion and for taking suitable prophylactic measures. There is the necessity of arranging for the treatment of these cases, almost all curable, so that they shall be sent back as combatants to their various units. With this object in view, the authorities have arranged for the treatment of all such patients in special departments—the Neurological or Neuro-psychiatric Centres of the interior or the army zones. This arrangement has given excellent results and, in spite of a few faults of detail, has rendered great service both to the patients themselves and to the community.

We have, then, finally to describe the method of treatment and cure of the psychoneuroses. Naturally, every neurologist has his own personal methods, which he prefers to all others because he gets the best results from them ; there are, of course, many methods of procedure, useful in certain cases and certain individuals, some of them rapid methods, some slow. Just as each patient modifies, to some degree, his psychoneurosis in his own fashion and often stamps it with his own individuality, so the physician himself will adopt a personal line in his therapeutic measures. It is, in fact, a question of the personal equation.

The uninitiated will, however, need some guide, some indication as to the course to pursue, when they find themselves face to face with a psychoneurosis, should no expert advice be obtainable. These patients are so often met with in the various hospital and Red Cross organizations in the army zone. A deaf-mute with symptoms of shock, a soldier with tremor, or a paralytic arrives at the field dressing-station, the field-ambulance or casualty clearing-station ; what is to be done with him ? Certain slight neuropathic disorders might be treated there and then to the patients' great advantage, provided that the medical officer of the regiment or divisional ambulance knows how to recognize them. Or a case of some functional nervous disorder arrives at one of the special departments : how should he be cared for ? For this purpose let us consider in order :

(1) The rôle of the physician.
(2) The rôle of the patient.
(3) The environment.
(4) The methods of treatment.

1. **The Physician.**—The medical officer plays the leading part ; on him depend to a large extent the results of treatment. Many failures are referable to the doctor. It is, indeed, common to see patients who carry their neuropathy, their tremor, their

spasmodic tic or nervous crises from hospital to hospital, and end by becoming "cured" when they have at last found "their master."

The physician who undertakes psychotherapy should have a strong will-power, for he has to acquire and exert over his patients the influence and ascendency which are essential if good results are to be obtained. In military medical work, a high degree of prestige is a valuable asset which will much facilitate this form of treatment, in which the physician has to play the double part of "confessor" and "educator." Convinced in his own mind of the curability of the disorder, he must communicate his conviction to the patient, entering into communion, into close psychical contact with him in order to impose upon him his own belief. This must be commenced as soon as the diagnosis has been established, during the first talk with the patient; little by little his confidence will be obtained, his mind mastered and finally conquered, if, as often happens, he shows a certain amount of resistance.

He must speak kindly but with authority, "with a hand of iron in a velvet glove." The probable success of the treatment can often be gauged from the first talk with the patient; when he leaves the doctor's room to go back to the ward, it is easy to judge from his behaviour during the interview whether a cure will be easy or difficult to obtain.

During the course of treatment great patience is needed to avoid losing one's temper, which might entail the dropping of a few unfortunate expressions which the patient will not be slow to utilize as a weapon of defence or offence. It is by no means always an easy task, and all those who have had experience of psychotherapy know that there are days and moments, when one is most successful in treating these patients. Therefore, when no success is obtained during the course of a protracted séance, the best plan is to give up for the time being and try again the following day,

on the pretext of giving the patient a rest. Care must, of course, be taken to isolate the patient in the meanwhile. This method of procedure by short, intermittent séances often succeeds; we adopt it ourselves, and on many occasions we have seen a paralytic leave the room still dragging his legs after him or a mute only speaking with great hesitation, but the following morning on our round found the patient declaring that he had completely recovered, which was actually the case.

The medical officer must also take care not to do or say anything that might give fresh suggestions (in the bad sense of the word) to the mind of his patient. We have already pointed out how common and dangerous such a practice is.

Lastly, care must be taken not to appear to suspect the patient of malingering, during the first few interviews at any rate, nor to be too ready to threaten disciplinary measures, court-martial, etc. These are outside the doctor's sphere and must be reserved for a special class of "patient," and only used as a last resort. We have never found them successful in the psychoneuroses.

The right method is for the doctor to see the patient face to face, to consider him as a neuropathic individual and to give him this impression, even though he believes him to be exaggerating or malingering, or under suspicion of either. He must bear in mind the difficulty of diagnosing malingering and only treat as such those who really deserve it. Fortunately it is the exception. To all others—those whose nervous systems have been deranged by the events of war, the "wounded in mind"—the physician must afford opportunity, power, energy or desire to recover, all of which they lack and are unable to attain by their own effort.

We will again insist on the necessity of making a methodical examination of the patient, even when the nature of the affection appears perfectly evident. This remark is far from superfluous; after seeing a

large number of neuropathic patients pass into one institution, one is very much inclined to be biassed by a first impression and to accept a diagnosis that has previously been made. A fresh and minute examination, including all the usual methods such as lumbar puncture, X-rays, etc., will correct any false diagnoses that have been made or reveal any association of hysteria with organic lesions ; further, it is a most valuable adjuvant in obtaining the confidence of the patient in the physician, who will confirm the curability of his disorder and promise a cure.

Lastly, great importance is attached to the psychological examination ; the patient should be encouraged to talk at some length, when his intellectual development may be gauged and his social status estimated, for the most valuable indications for treatment are thus acquired ; an individual of low intellectual development will need methods of treatment different from those of an intelligent and well-educated person.

2. **The Patient.**—All the psychoneuroses are not equally amenable to psychotherapy.

There is a group of patients—the real psychopaths (confusional, melancholic, psychasthenic states)—that must be set apart as belonging more definitely to psychiatry and requiring special treatment, such as prolonged rest, hydrotherapy, etc. Some of these may, of course, recover more quickly.

The others, *i.e.* the psychoneuroses of hysterical or pithiatic type, come within the scope of psychotherapy in the strict sense of the word.

Certain inherent conditions of the patient and the nature of his complaint must be taken into consideration. A new patient, not yet examined or treated, is much more easily cured than one in which the symptoms date back some time. The latter, a confirmed neuropath, often requires special methods of treatment which we shall mention later. The degree

of curability is thus inversely proportional to the duration of the disorder. *Hence the advantage to be gained by treating the patient as soon as possible after the trauma.* This is an axiom now universally admitted.

There are other factors which influence the treatment and should be taken into account.

The nature of the psychoneurosis is of importance : contractures are more persistent than paralysis, tremor and spasmodic tic are more obstinate than other results of shock such as deaf-mutism, etc.

The psychological state of the patient also exerts an influence. It is obvious, for example, that the most telling argument will have little effect on an individual of poor intellect.

Lastly, much difficulty will be encountered in the treatment of patients who even before the war had exhibited nervous phenomena. The *pre-war psychoneuroses* are more intractable than the *psychoneuroses of the war* ; nevertheless, results can be obtained which will render the patient fit for some service.

3. **The Environment.**—We have seen that the personality of the physician is of the greatest importance as regards the results of treatment, by reason of the moral authority which he exercises ; we have also seen that the psychical state of the patient, the duration and nature of the disorder play an influential part. There is a third factor, no less important—the surroundings in which the psychoneuroses are cared for and treated.

This is clearly shown by the fact that the hospitals near the fighting-front (neurological centres near the front) prove eminently suitable from this point of view. Their only relative degree of comfort, their strict military discipline, their proximity to the front, their remoteness and their inaccessibility to friends and relations all render them specially suitable for this form of treatment and ensure much

easier and quicker cure than in the interior. Two years' observations have fully confirmed this fact, which is now admitted by all neurologists. It is regrettable that the authorities have not been able to arrange a more effective sorting-out of these cases; a large number, especially at the time of offensive operations, still get sent to hospitals in the interior for months on end.

We do not deny that these patients can be cured in the various neurological centres in the interior, but with what difficulty and how slowly! Here, the duration of treatment is reckoned in weeks or months, while at the front it is obtained in a few hours or a day.

But the question of situation—near the front or in the interior—though important, is not the only one. A suitable atmosphere and proper surroundings must be created, and this should be particularly easy under military régime.

The discipline, both general and medical, should be severe but just; the personnel, attendants and nurses alike must be well trained in the right behaviour and bearing towards this class of patient.

On admission the patients will realize that they are going to be in the hands of a physician who can cure them; but that certain noisy nervous manifestations, such as hysterical convulsive attacks, are forbidden, under pain of a more or less prolonged confinement in a separate room, and that every chronic neuropath is kept strictly in bed on a milk-diet until the symptoms improve. When once this routine is established, cures will follow one another with great rapidity.

It need not be thought that the creation of such an atmosphere and routine is a matter of great difficulty. It can be done in two or three weeks; we have effected it several times in the course of our work in various departments of the neurological service.

4. **The Methods.**—We must now discuss the treatment of the psychoneuroses. It consists of a psychotherapy which is methodical, firm, and often even imperative; it includes more or less prolonged conversations, assisted by isolation, application of the faradic current, and re-education.

We may term it the *psycho-electric and re-educative method,* and it may be divided into four stages:

1*st Stage. Persuasive Conversations.*—The patient has, if possible, been examined on the day of admission. If not, he will have been previously isolated. When the case has been diagnosed, one tries to explain to the patient the nature of the disorder from which he is suffering or believes himself to be suffering (the result of a mental or physical shock, fear, fatigue, etc.), to show him how it has arisen, to reassure him as to its gravity, and lastly to promise him a cure. All this must be done simply, quietly, and kindly but firmly. If opportunity arises, one can encourage him to chat to any patients who have recovered and have not yet been discharged. This "benign contagion" from patient to patient often works well in cases of deaf-mutism and nervous crises, for example.

During the course of this first talk with the patient use is made of methodical suggestion, in the waking state, of course; this is all the easier to carry out when one has to deal with patients on the lower intellectual levels.

This séance of psycho-therapeutic conversation should take place in the doctor's consulting-room and not in the ward.

At the end of the conversation one should endeavour to get the patient to say that he really wants to get well and that he will submit to any methods of treatment necessary to that end. The promise is made on oath, and from the inclusion of any conditional clauses or evasions, one is able there and

then to draw conclusions as to the ease or difficulty with which the patient is going to be cured.

The next stage of treatment may be begun on the same day and electrical methods employed. But it is better to proceed methodically and to postpone the latter till the following day, or better still till several days later, during which time the patient is kept in strict isolation.

2nd Stage. Isolation.—The patient is then put in a separate room and kept in bed on a strict milk-diet. Save for rare exceptions, isolation is a valuable or even indispensable aid to psychotherapy and has been a classical method of treatment since the days of Weir Mitchell; we need not discuss it further.

By proceeding in this way by successive steps, isolating the patient for a few days and giving him electrical treatment, several objects are achieved :

(*a*) The effects of suggestion are reinforced. They are, of course, maintained by short talks with the patient when the daily rounds are made.

(*b*) The patient is left alone with his thoughts and has an opportunity of thinking over the promises he has made ; it often happens that he will eventually beg for the electrical treatment which at first he refused.

(*c*) The period of observation is lengthened, allowing time to complete the diagnosis.

Isolation is useful not only as a method of treatment, but it is also a prophylactic measure avoiding all chance of " contagion." There are two degrees : *comfortable isolation* in a separate room, which is adequate in most cases, and *rigorous isolation* in a cell with milk or bread and water diet, to which recourse must sometimes be had.

Often during this second stage of treatment spontaneous recovery takes place. Claudication of long duration, tremor, or deaf-mutism may disappear as if by magic before electrical treatment has been

commenced. We have seen many examples of this. Now we pass on to the next stage.

3rd Stage. Faradization.—Electrical treatment is carried out by the physician alone, only the attendants necessary to support the patient being present.

Position of the Patient.—The patient lies absolutely naked on the bed, where he is first treated in the recumbent position, especially in motor affections of the lower limbs. Afterwards he is treated sitting down, then standing, walking, running, etc.

Apparatus.—The apparatus for faradization supplied to the medical services is the type used; the dry cells may be advantageously replaced by Leclanché batteries, which are connected up in series. A spool of fine wire is used, and as stimulators at first pads, then revolving cylinders, then a metal brush. The current is at first feeble and then gradually increased; the poles are first applied to the affected parts and then, if necessary, to specially sensitive parts of the skin surface (ears, neck, lips, sole of the foot, perineum, scrotum).

Care must be taken to proceed gently at first, pointing out to the patient any contraction of so-called paralysed muscles, comparing them with those of the unaffected limb; then, if needs be, the strength of the current is increased and more energetic measures used. This is the rapid method, indicated in the vast majority of cases, especially at the front. In cases seen early it almost always gives immediate results, and is of special value in psycho-sensorial and sensory disorders due to shock, nervous crises, and most of the psycho-motor disorders.

It really depends on producing a kind of "crisis," which we should try to obtain at the first séance. The latter often has to be continued for some hours, until the patient is finally "mastered." But if this crisis is much delayed and the patient shows obvious signs of fatigue, it is better to suspend operations and continue the following day. This often happens

in affections of long standing ; some patients require two or three séances, some even more than this.

Hydrotherapy.—A cold douche, either as a shower or an unbroken jet of water, may be used instead of faradization to obtain the crisis. We have used it with success in hospitals near the fighting-front in the treatment of recent cases of shock. The same may be said of certain other methods, such as slightly painful subcutaneous injections (ether), which may be used, for want of other means, in advanced posts in the firing-line (Boisseau).

4th Stage. Physical and Psychical Re-education.—This is an important branch of psychotherapy, particularly in cases of long-standing disorders. Most neuropaths (especially in the centres in the interior), when once the crisis has been obtained, need re-education by systematic exercises directed towards the re-establishment of the lost functions—motor re-education, helped by massage and passive movements —in short, the various forms of physiotherapy. This re-education should be carried out by special assistants or head nurses ; it should always be accompanied by psychotherapy and, if needs be, by further electrical treatment.

Re-educative methods of treatment alone, not preceded by faradization, are employed by certain neurologists. Satisfactory results may be obtained, but they are slow and tedious. Adequate enough in time of peace, they by no means meet the necessities of war ; rapid methods are absolutely necessary when one is faced with a large number of such patients, who soon fill up the hospitals and make them literal " culture-media." [1]

[1] At the Neurological Centre of District IX., Clovis Vincent has instituted a method of intensive re-education for old hysterical cases, which is carried out in three stages :

(1) *Stage of crisis and intensive re-education.*—This is the stage called "torpillage" by the poilu, during which the patient's

After-Treatment.—After recovery has taken place, the patient should be kept for a time under observation before being sent back to his regiment or to the depôt. In the course of the daily round he should be watched and assured of the permanence of his recovery. Unfortunately it is not always possible at the front, owing to lack of accommodation, to keep these hysterical cases for an adequate length of time. Premature transference to hospitals in the interior or too early sick-leave are frequent causes of relapse. In hospitals in the interior, on the other hand, these drawbacks are not met with ; the patient can be retained, and trained in the centres for re-education.

Results.—In closing, we can say, without exaggeration, that almost all the psychoneuroses of war of hysterical type can be cured by well-conducted psychotherapy.

inertia is overcome, a stage of forcible re-education, effected by the galvanic current. It includes

A. *Direct Re-education,* in which the patient is compelled to make a movement or perform an action of which he says he is incapable ; and *Indirect Re-education,* in which he is led to the desired goal mechanically and without the intervention of his own will (running, for example, to restore the normal automatism of the lower limbs).

B. *Application of the Galvanic Current,* rising to a strength of 30 or 35 milliampères, employed for a short time only at repeated intervals during treatment.

(2) *Stage of fixation of the progress obtained,* in which various movements, walking exercises, etc., are carried out to maintain the cure.

(3) *Stage of training,* carried out by special assistants over a prolonged period, under the doctor's supervision, before the patient is sent back to active service, definitely cured.

This method has given excellent results, but it applies to a special class of neuropaths—old hysterical cases, which for months, or may be for over a year, have been sent from one hospital to another, and who have never had correct treatment since the onset of the disorder. At the front there are extremely few cases suitable for this method.

With regard to *failures*, of which there are unfortunately many more than there should be, it must be admitted that these are generally due to some fault in treatment: unfavourable surroundings, initial mistakes in diagnosis, too slow methods, lack of interest or time on the part of the physician.

More rarely they are due to the psychical state of the patient himself: obstinacy, ill-will, refusal of treatment.

In one of the Army Neurological Centres during a period of six months we have had a percentage of 98-99 per cent. of recoveries (Roussy and Boisseau).

In one of the hospitals in the interior, specially organized for this treatment, only 2-3 per cent. of failures have been recorded (Clovis Vincent).

The cure of a psycho-neuropath really consists of a mental contest, resulting in the victory of the physician. This, in conclusion, is the secret of psychotherapy.

THE PSYCHONEUROSES AND MILITARY DECISIONS

We have still to consider the question of the military decisions that have to be made as regards cured and uncured psychoneuroses, a problem that often presents great difficulties of solution.

The heads of the various neurological centres have frequently drawn the attention of the military authorities to this matter and have requested instructions which will lead to a more uniform medico-military procedure.

The Paris Neurological Society had this subject under discussion at their plenary sitting of last December ; we shall give an account of the decisions arrived at.

We must examine the various questions that arise, both in the Neurological Centres at the front and in the interior, according to the two classes of patient :

Those in which the neuropathic element predominates (hysterical or pithiatic), and

Those in which the psychical element predominates.

Neurological Centres in the Army Zone.— As regards the hysterical type of case, we have seen that in principle all of these must be regarded as susceptible of cure. In fact, personal experience shows that this occurs in the large majority of cases. Failures only occur with great rarity and cannot be discussed here.

The only questions that arise are these :

(1) How soon after recovery from a psychoneurosis shall the patient be sent back to active service ?

(2) Should he be given a week's leave of absence or leave for convalescence ?

It is not such an easy matter as it may appear at first sight to decide on the right time to send the cured patient back to service. Too often, indeed, as Grasset very rightly points out, a patient is rapidly "cured" and then sent straight back to his unit. As soon as he gets back to the trenches, his symptoms reappear and again necessitate evacuation. We ourselves made this mistake before we realized the situation. And even to-day, we often receive cases sent on from neurological centres of neighbouring army units, in whom consolidation of the cure does not seem to have been properly effected.

A seven days' convalescent leave for every patient who has been ten days in a hospital is an army regulation, and, in spite of the resolutions passed by the Congress of Neurologists at Doullens in January 1916, the refusal of this leave is, in certain army groups, considered as a disciplinary measure which necessitates the exchange of various documents and reports. This is a great mistake, and it is to be hoped that greater latitude will be allowed to the heads of the army neurological centres. It is desirable that this leave should only be granted for special reasons to patients who have shown goodwill and intentions during the course of treatment. Too often one is biassed by a very natural sentiment and led to consent, only to have to regret the decision later. We can quote two recent instances of this : a hysterical patient, after having been under our care for six weeks without a nervous crisis—though he had had them daily for several months previous to this—was given leave to go to Corsica and had an attack on the boat ; another patient, distinguished with the Legion of Honour, having been cured in a few moments of deaf-mutism due to shock, failed to rejoin his unit on the expiration of his leave of absence.

With regard to *prolonged convalescent leave of absence*, we think *a fortiori* that it should only be granted in exceptional cases in the army zone. A

hysterical patient derives no benefit from a long stay at home amongst his friends.

Lastly, cases of psychoneurosis should not, as often takes place, be transferred to centres in the interior except under exceptional circumstances. We need not again dwell on the necessity of exercising the greatest care in sorting out this class of patient with accuracy in the casualty clearing-stations and sorting-stations. It goes without saying that purely military reasons may suddenly necessitate the evacuation *en bloc* of a hospital containing neuropathic patients under treatment.

In the case of patients in whom the psychical element predominates—neurasthenic conditions, traumatic neurosis, confusional states, sequelæ of shock, etc.—the decisions that must be made are different. A minority of these patients recover quickly and can be treated at the rear, but here again a too early return to the front is often followed by relapse. The majority need, as we have already seen, transference to centres in the interior, as they require a prolonged period of rest and convalescent leave.

Neurological Centres of the Interior.—In these branches of the medical service, the problems that arise are more numerous and more complex; the special class of psychoneuroses with long-standing symptoms, the large number of cases that have to be dealt with, and the depletion of the medical and nursing staff all go to render the conditions less favourable for psychotherapy.

As regards the cured cases of psychoneurosis of the hysterical type, we can make the same reservations as to return to service or convalescent leave.

What should be done with uncured patients, obstinate and chronic cases of hysteria? This is the question that so often faces the heads of the neurological centres and of which the Under-Secre-

tary of State for the Medical Services has recently suggested a solution.

As Grasset has put it, "They are patients who cannot be sent back to the depôt (because there is no one to look after them) and who cannot be sent home to recover their health (because they are not convalescents and return from home in a worse condition than before); they cannot be discharged, because on October 21st, 1915, the Neurological Society decided that '*at the present time, under no circumstances whatever, must any soldier suffering from a psychoneurosis be brought before a medical board for any question of discharge,*' and since, on November 6th, 1915, the Under-Secretary of State for the Medical Services wrote to the President of the Society to intimate his approval of this decision."

What, then, is to be done with such cases, the number of whom is increasing daily and who constitute both a danger of contagion and a bad example to their fellow-patients? At the last meeting of the administrators of the neuro-psychiatric centres, held at Val-de-Grâce under the presidency of the Under-Secretary of State, the following measure was proposed and adopted:—Old and obstinate cases of hysteria are to be transferred in small batches, under arrangements made by the department, to the neurological centres in the army zone, some of which will be organized in view of this special treatment. We are in entire agreement with this decision, which, to judge from our personal experience, will certainly lead to a diminution in the number of these "undesirables" and the restoration of a large number of them to the army.

Cases of Psychoneurosis and the Discharge Boards.—In spite of the resolutions passed by the Neurological Society in 1915 with regard to the discharge of uncured cases of hysteria, there are many different medico-military standards applied to the psychoneuroses in the various districts. Far

too common are the patients of this class that have been, and still are, granted temporary discharge, with or without pay, as a sort of last resource. This is an extremely bad example to other patients under treatment and compromises the results. The Paris Neurological Society therefore devoted its meeting of December 15th last to the question of the Neuroses and Psychoneuroses of War and the Methods of Dealing with Them.

The following suggestions were proposed by Babinski and adopted :

(1) For purely hysterical or pithiatic affections—neither discharge nor pension.

(2) For pithiatic affections associated with organic or physiopathic disorders—no account is to be taken of the hysterical manifestations in estimating the degree of incapacity.

(3) For the physiopathic disorders ("reflex" nervous disorders) following trauma in the war and uncured after prolonged treatment—auxiliary service or temporary discharge with a pension of 20-50 per cent.

(4) For characteristic neurasthenic conditions, not accompanied by organic disorders or mental symptoms—no pay, very exceptionally discharge, generally auxiliary service.

In conclusion, may we raise a last question that of the interpretation by the discharge-boards of the decisions arrived at by the doctors, particularly the specialists. In spite of the many and frequent protests that have been raised by the medical administrators of general and specialized departments alike, the contest still goes on. The discharge-boards persist in taking the least possible notice of the decisions of the medical officers, who have often taken a long enough time in making quite certain of an accurate diagnosis.

We cannot do better than quote the words of Grasset's report, which every medical man who is

conscious of his responsibility will certainly approve:

"The following is the actual text of order No. 309, Ci 7 C.C.M., Nov. 5th, 1916, on the powers and province of general physicians and *specialists* in the making-up of medico-legal reports: 'The duty of general physicians and particularly of *specialists*, in making up their reports, is to furnish the most complete anatomical and clinical information in order to allow the only legal authorities—the expert medical advisers to the boards—*to express an opinion* or to arrive at decisions as to the legitimate course to be adopted with regard to the case in question. The physician should therefore *abstain from making any personal estimate as to the degree of incapacity or any suggestion as to a definite decision to influence the board,* save in exceptional cases, when in certain circumstances the board or its expert medical advisers may request their advice as consultants and in cases where the physicians are called for a second opinion by government order . . .'

"This complete suppression of all element of specialism is based solely on the objection that the patient knows the suggestion or decision of the competent specialists; the same regulation discovers an easy remedy for this, by demanding, in the exceptional circumstances referred to above, that the information be forwarded in a sealed package."

The Discharge Boards, we are informed, have supreme jurisdiction in the matter. But regulations are not unalterable, and it is not very revolutionary, in view of present events, which have upset all the old order of things, to urge the repeal of obsolete measures, which may have been adequate in time of peace but which no longer correspond to the needs of the times through which we are now passing.

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